THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—40TH YEAR SYDNEY, SATURDAY, AUGUST 8, 1953 No. 6

Table of Contents.

[The Whole of the Literary Matter in THE MEDICAL JOURNAL OF AUSTRALIA is Copyright.]

ORIGINAL ARTICLES— Page.

Physiological Principles in the Management of Fluid and Electrolyte Problems in Infancy and Childhood, by W. B. Macdonald 197
Fluid and Electrolyte Problems in the Newborn, by Kate Campbell 201
The Pathological Physiology, Assessment and Management of "Dehydration" in Entero-Colitis, by Howard Williams 203
Fluid and Electrolyte Problems in Infants and Children: Intestinal Obstruction, by E. S. Stuckey 205
The Minor Epilepsies, by D. G. Hamilton 207
Vitamin D Resistant Rickets, by Douglas Galbraith 208
Congenital or Neo-Natal Thrombocytopenic Purpura, by S. E. L. Stening 210
Recent Trends in the Diagnosis and Treatment of Cœliac Disease, by Charlotte M. Anderson .. 211
Urinary Excretion of Mercury in Pink Disease: Preliminary Report, by F. W. Clements 213
Poliomyelitis Virus in Tissue Culture, by J. R. Thayer 215
Coxsackie Virus Infections in Australia, with Special Reference to Their Epidemiology, by N. F. Stanley 216

REPORTS OF CASES—
Idiopathic Hypoparathyroidism, by Felix Arden .. 217

REVIEWS—
Infant Feeding 219
Physiologic Therapy for Obstructive Vascular Disease 220

BOOKS RECEIVED 220

LEADING ARTICLES—
Pædiatrics 221

CURRENT COMMENT—
Migraine in Children 222
Poliomyelitis in French Oceania 223
Treatment of Whooping-Cough with Antibiotics .. 223

ABSTRACTS FROM MEDICAL LITERATURE—
Pædiatrics 224

MEDICAL SOCIETIES— Page.
Australian Pædiatric Association 226

OUT OF THE PAST 241

CORRESPONDENCE—
Choledochoduodenostomy 241
The Treatment of Chronic Rheumatism with "Butazolidin" 241
Congenital Hypertrophic Pulmonary Stenosis in Aboriginal Children 242
Exchange Transfusion and Transfusion in Infants and Young Children 242
Another Record of a Human Diphyllobothriid in Australia 242

POST-GRADUATE WORK—
The Melbourne Permanent Post-Graduate Committee 242

ROYAL AUSTRALASIAN COLLEGE OF SURGEONS—
Primary Examination for the F.R.A.C.S. 242
Final Fellowship Examination 243

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA 243

NAVAL, MILITARY AND AIR FORCE—
Appointments 243

MEDICAL PRACTICE—
National Health (Pensioners' Medical Services Committees of Inquiry) Regulations 244

AUSTRALIAN MEDICAL BOARD PROCEEDINGS—
Queensland 244

DEATHS 244

DIARY FOR THE MONTH 244

MEDICAL APPOINTMENTS: IMPORTANT NOTICE .. 244

EDITORIAL NOTICES 244

PHYSIOLOGICAL PRINCIPLES IN THE MANAGEMENT OF FLUID AND ELECTROLYTE PROBLEMS IN INFANCY AND CHILDHOOD.[1]

By W. B. MACDONALD,
Children's Hospital, Melbourne.

IN all branches of medicine an appreciation of underlying physiological disorders is necessary, and this is particularly evident in diseases with which are associated disorders of fluid and electrolyte metabolism. The subject incorporates physico-chemical principles which are the basis of metabolism and the maintenance of life, and an unsound knowledge will sooner or later lead to serious maltreatment. Each case calls for individual assessment on physiological grounds and the avoidance of rigidity in management.

[1] Part of a symposium on "Fluid and Electrolyte Problems", held at the annual meeting of the Australian Pædiatric Association, Canberra, April 10, 1953.

Definition of Terms.

Before proceeding further, it will be necessary to define certain units of measurement in fluid and electrolyte work. The unit of chemical activity of an electrolyte is the milliequivalent and is expressed in solution as milliequivalents per litre. The expression of an electrolyte in terms of milligrammes *per centum* is meaningless, as chemical activity has no relation to "weight". By converting all electrolytes to a common unit of chemical combining power, the number of chemically active particles in a solution can be read directly. The calculation of milliequivalents per litre from milligrammes *per centum* is: Milliequivalents per litre =

$$\frac{\text{milligrammes per centum} \times \text{valency} \times 10}{\text{atomic weight}}$$

The combining power is proportional to valency. To make an analogy which is by no means complete, one expresses the power of an automobile in terms of horse-power and not in terms of the weight of the vehicle. To persist with the unit of measurement of milligrammes *per centum* reveals a lack of understanding of the subject. The expression of milliequivalents per litre has the added

advantage that the total cations must equal total anions, thus providing a check on results.

Ionic concentration of a solution is expressed as milliosmols per litre, the calculation of which is similar to that for milliequivalents per litre, except that as chemical activity is of no significance in assessing osmolarity, valency is disregarded.

The smooth working of cell activity, and hence metabolism and life, requires the maintenance of steady physicochemical states both inside cells and in the environment of the cells, the extracellular fluid. There is an optimal range of values, deviation from which interferes with cell function. Homœostasis, a well-known term, is defined as "the maintenance of steady states in the organism by coordinated physiological processes involving the activities of numerous systems". Homœostasis involves many factors which are outside the scope of this presentation, but restricted to fluid and electrolyte metabolism, it requires the maintenance of an optimal range of values in the following components: volume, ionic concentration, pH, electrolyte pattern, of body compartments, both intracellular and extracellular. To this end the organism's regulators act, and to assist them in homœostasis is the aim of the fluid therapist.

Normal Values of Components.

The known normal values of these components in intracellular and extracellular fluid have been summarized.

Volume.—Gamble states that 70% of body weight is water, 5% of body weight being plasma and 15% interstitial fluid, but Darrow considers that 25% to 30% of body weight in infants is extracellular fluid. Results of other workers indicate that total body water in adults may be lower than the figures commonly accepted.

Ionic Concentration.—Ionic concentration is 310 milliosmols per litre in extracellular fluid, but intracellular osmotic concentration has not been measured. It is probably equal to that in extracellular fluid, although there is some difference of opinion. The greater number of milliequivalents per litre in cell substance does not preclude isotonicity, as the valencies of cellular electrolytes are higher.

The pH Value.—The pH is 7·35 to 7·45.

Electrolyte Pattern.—In the case of cell substance, electrolyte pattern varies slightly with different cells and is less accurately obtainable. The important position of potassium, magnesium, phosphate and protein in cell substance, in contrast to sodium, chloride, and bicarbonate in extracellular fluid can be seen (Figure I). Most intracellular phosphate is in organic form in equilibrium with a small concentration of inorganic phosphate, and it will be remembered how important the organic phosphates are in the composition of enzymes. Potassium, the main intracellular cation, has been shown experimentally to have a predilection for intracellular substance far in excess of sodium, distinct from any factor of membrane permeability.

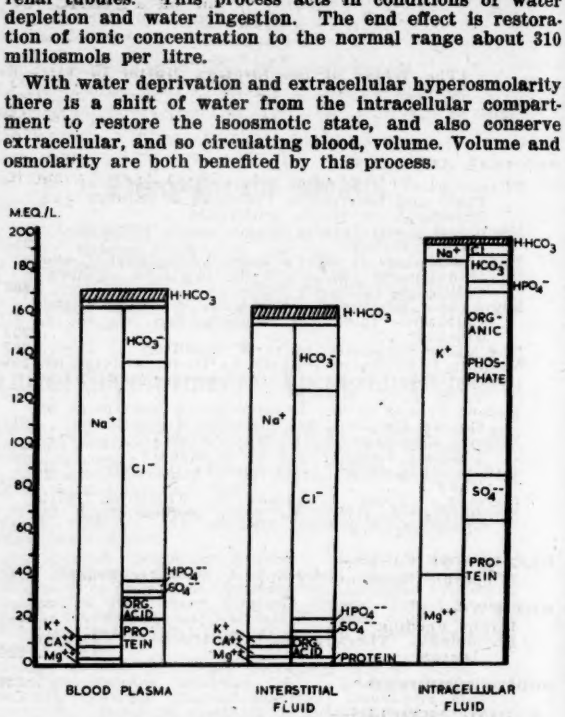
To maintain normal values of the intracellular physicochemical components of volume, ionic concentration, pH and electrolyte pattern, a steady state must exist in the same components of extracellular fluid, which represents the environment of the cells (the *milieu intérieur* of Claude Bernard). The mechanism of maintaining this depends on the coordinated activities of receptor organs which respond to environmental changes, endocrine glands and their hormones and effector organs, the most prominent being the kidneys and lungs. In abnormal conditions sometimes the optimum cannot be maintained in all chemico-physical states making up the *milieu intérieur*, and speaking teleologically, one has to be sacrificed as it were to the welfare of another.

Regulatory Mechanisms.

Consider in turn the mechanisms which act for the preservation of the steady state in these components.

Ionic Concentration.—The maintenance of ionic concentration in many ways is best understood and will be considered first. The receptor organs are the nuclei of the hypothalamus (the supraoptic and paraventricular nuclei). The endocrine gland is the posterior pituitary lobe connected to the hypothalamic nuclei by the supraopticohypophyseal tract. The hormone is pitressin and the effector organ the distal renal tubules. In states of hyperosmolarity there is increased secretion of anti-diuretic hormone, and in states of hypoosmolarity an inhibition of secretion with reduction or lack of reabsorption of water from the distal renal tubules. This process acts in conditions of water depletion and water ingestion. The end effect is restoration of ionic concentration to the normal range about 310 milliosmols per litre.

With water deprivation and extracellular hyperosmolarity there is a shift of water from the intracellular compartment to restore the isoosmotic state, and also conserve extracellular, and so circulating blood, volume. Volume and osmolarity are both benefited by this process.



ELECTROLYTE PATTERN IN BODY FLUID COMPARTMENTS

FIGURE I.
Electrolyte pattern in body fluid compartments.

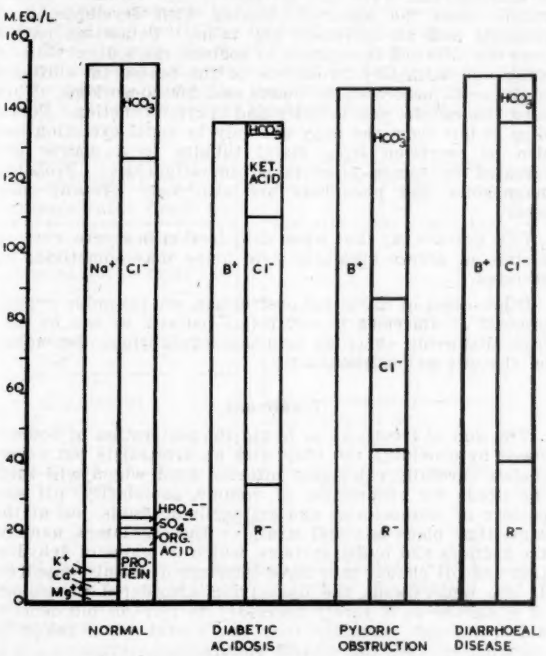
With water ingestion and good renal function there is no danger of intracellular water shift and water intoxication, except in very young infants.

After water ingestion and water deprivation, as mentioned, the maintenance of an isoosmotic state aids the attainment of normal extracellular volume.

However, when the predominantly extracellular electrolyte sodium is gained or lost, the converse occurs.

Much work has been done recently on sodium retention and œdema, and renal excretion of sodium, but my comments will be confined to sodium depletion which is common and relevant to many disorders of infancy and childhood. In disorders which cause electrolyte depletion without a corresponding water depletion, exemplified by some cases of infantile diarrhœa, there is reduction of electrolyte concentration, which without the addition of electrolytes can be restored to isotonic levels only by excretion of water. This of necessity reduces extracellular and blood volumes. The "body" has to decide between reduction of volume in favour of the maintenance of ionic concentration and the contrary. Excretion of water, being the common mechanism in the normal, takes precedence initially, but when circulating blood volume becomes embarrassingly reduced, a compromise occurs and concentration falls so as to maintain extracellular and hence blood volumes. This is seen in Addisonian states, and more commonly in infantile diarrhœa in which electrolyte replacement therapy has been

poor. The mechanism which results in the precedence of volume over concentration is not completely known. The efficient renal conservation of sodium is probably due to a dual effect of decreased glomerular filtration rate, at least in infancy, and increased sodium reabsorption from renal tubules governed by the activity of adrenal corticoids. The fall in glomerular filtration rate is not always sufficient to explain water retention, however, and this could be due to the effect of reduced blood volume on a volume controlling mechanism quite distinct from antidiuretic hormone, whose activity in this case does not conserve volume. Despite all efforts to maintain circulating blood volume, there is a limit to the compromise and when hypoosmolarity becomes extreme, circulatory failure occurs, renal function is impaired and water conservation fails, even to the extent of resulting in a hyperosmotic state, according to Kerpel Fronius. The importance of maintaining an adequate circulation need hardly be stressed. Without it,



THE PLASMA ELECTROLYTE PATTERN IN CERTAIN DISEASES

FIGURE II.

The plasma electrolyte pattern in certain diseases. B^+ = total cations. R^- = the sum of the remaining anions not specifically indicated in the anion columns.

oxygen carriage is greatly impaired and metabolism slowed down ultimately to a halt.

The pH Value.—The maintenance of a pH 7·35 to 7·45 in the *milieu intérieur* is of the utmost importance. It is maintained by the action of various buffer systems, bicarbonate, phosphate, intracellular proteins and hæmoglobin, renal excretion of fixed acids and renal tubular ammonia production, details of which will not be discussed.

Low pH resulting, for example, from diarrhœa or diabetic acidosis causes hydrolysis of the intracellular substance, especially the organic phosphates. There develops an inevitable leakage of potassium, magnesium and inorganic phosphates from the cells, and the membrane barrier does little to prevent this. Inevitable renal excretion occurs with total body deprivation and disorganization of cell processes which persists despite replacement of extracellular electrolytes.

In alkalotic states with an elevation of pH, seen in pyloric stenosis, a state quite as hazardous can develop. In this situation, cell potassium is lost and its place is taken by sodium. Not only does renal excretion of potassium occur, but it is actually secreted by the distal renal tubules according to Berliner. Darrow first pointed out that the alkalotic state cannot be corrected without potassium, and potassium replacement into the cells cannot be managed without correction of the alkalosis. Again there must be great disorganization of cell function even in the presence of a normal circulating blood volume and ionic concentration.

Another hazard of alkalosis is reduction of ionized calcium and the development of tetany. The importance of maintaining a normal pH can therefore be appreciated.

Electrolyte Pattern.—Under normal conditions the electrolyte pattern of intracellular and extracellular fluid remains within a fairly constant range. Figure I demonstrates the pattern in vascular, interstitial and cellular compartments. The manner in which stresses affecting acid-base equilibrium alter pattern has been briefly mentioned, and, practically speaking, in disease states one does not occur without the other. However, apart from pH control, there are specific effects due to electrolytes present in concentrations outside the normal range in both cellular and extracellular compartments. Continued stress usually associated with acidosis or alkalosis has been shown to cause depletion of potassium, magnesium and phosphates, and replacement of potassium by sodium in the cells in alkalotic states. Altered electrolyte pattern in extracellular fluid is corrected by healthy kidneys, in many cases at the expense of total ionic concentration, as demonstrated by Denton and co-workers. When renal failure occurs, gross pattern distortion develops and specific electrolyte effects are seen. It is most important to realize that, in states of severe dehydration with circulatory failure and impaired renal blood supply, renal function may be so affected that pattern control is lost, and some ions by specific effects in both high and low concentrations may cause severe symptoms and even death. There is probably a fine relationship between most electrolytes so that alteration in concentration of one may reciprocally affect another, and to attribute clinical effects to one particular electrolyte might be greatly over-simplifying matters. Serum biochemical findings will not accurately reflect intracellular electrolyte depletion. The extracellular fluid may merely act as a medium for transfer of electrolytes such as potassium from the cells to the urine.

Specific Effects of Electrolytes.

The more prominent specific effects of electrolytes, with the proviso that some may be due to reciprocal effects on others, will be briefly mentioned.

Potassium.—Depletion of potassium causes mental depression and muscular paresis, shown by apathy, stupor, abdominal distension, nausea, vomiting, ileus, diaphragmatic and intercostal paresis, rapid breathing, dyspnœa, skeletal paresis, loss of deep tendon reflexes, spoiling and necrosis of myocardium with cardiac dilatation leading to congestive cardiac failure. Electrocardiographic changes may occur—increased *Q-T* interval, low, rounded, prolonged *T* waves which may become inverted, depression of *S-T* segment, appearance of *U* waves, auriculo-ventricular block.

Increased potassium concentration in the extracellular fluid may cause mental confusion, apathy, bradycardia, irregular rhythm, extrasystoles, cardiac arrest. Electrocardiographic changes commencing at 6·5 to 8·0 milliequivalents per litre are peaked *T* waves, splaying distorted *QRS* complex, bundle branch block, absence of *P* waves, total arrhythmia leading to cardiac arrest.

Calcium.—Tetany may occur in conditions of low calcium concentration, but is more commonly seen in alkalotic states with decreased ionized calcium content. It may be precipitated by a sudden increase in extracellular potassium concentration or in renal failure with an increase in phosphate concentration because of reciprocal potassium, calcium and potassium phosphate relationships respectively.

It is well to remember that tetany in infants may be first clinically manifested by convulsions.

An increase of calcium concentration in extracellular fluid usually has little clinical effect, but when a sudden increase results from unrestrained calcium infusion in the presence of poor renal function, tachycardia, extrasystoles and even ventricular fibrillation may occur.

Magnesium.—Magnesium is an important component of cellular enzyme systems and may be depleted under circumstances similar to those causing potassium depletion. There is no clinical syndrome of magnesium depletion described, but some features attributed to potassium depletion may be due to this. Magnesium in low concentrations has been given intravenously in the treatment of diabetic acidosis in adults.

High extracellular magnesium concentration may cause mental depression, anæsthesia, muscular paralysis and death, and because of the low concentration existing normally in the extracellular fluid its use would be hazardous in the treatment of small infants.

Phosphates.—Phosphates in organic form comprise the main anions of intracellular substance and must be considered of prime importance. The phosphate buffer system is important in the renal regulation of pH.

Proteins.—Proteins are important in the constitution of cellular substance, and as buffers, but will not be further discussed.

Chloride.—Much has been made of the importance of chloride ion and it has become a clinical *cliché* to talk of chloride levels in plasma and urine. However, as far as is known, it has no specific action and is important because of its concentration in extracellular fluid, the part it plays with bicarbonate in the maintenance of acid-base equilibrium, and its relationship to sodium in extracellular fluid as the most prominent anion. In certain abnormal states in which depletion of sodium, more than chloride, occurs, urinary chloride may be present in some concentration despite the fact that plasma chloride level is well below normal. This has been shown by Denton and co-workers and would apply to most cases of infective diarrhœa in infancy. As a general statement, urinary chloride estimations quite apart from technical errors in ward testing can be interpreted only in relation to the overall physiological picture, which unfortunately is rarely appreciated.

The importance of sodium in relation to the maintenance of extracellular volume, and of bicarbonate in relation to acid-base equilibrium has been mentioned.

Stresses Caused by Certain Diseases.—On the basis of homœostasis in relation to fluid and electrolytes, consider very briefly the stresses caused by certain disease processes, and the physiological reactions to them.

Infective diarrhœa represents a sodium subtraction acidosis. In acute cases, water loss may exceed electrolyte loss, because of vomiting, failure of intake and sweating, and hyperosmolarity develops in the extracellular fluid. This results in a shift of water from cells. Intracellular electrolyte depletion is slight, but the volumes of both compartments fall.

With further diarrhœa, sodium loss persists and contraction of extracellular space continues. If water intake is unrestricted, hypoosmolarity results. The pattern of extracellular fluid is distorted, sodium and bicarbonate decrease in concentration, but chloride falls slightly if at all. Starvation may extend the organic acid concentration, pH falls, and a potassium, phosphate, and probably magnesium shift from the cells which starvation has initiated, becomes accelerated, leading to total depletion by renal excretion and loss from the stools.

Diabetic acidosis represents an anion addition acidosis. Initially the urinary loss from osmotic glycosuria can be easily replaced orally, but as keto acids increase and the renal ability to excrete fixed acids and produce ammonia becomes exhausted, unavoidable loss of sodium occurs. Anorexia and later vomiting precipitate serious dehydration, the extracellular electrolyte concentration depending on relative sodium and water loss. The pH falls and hydrolysis of cell substance occurs with a shift of potassium, magnesium and phosphate to the extracellular fluid. If dehydration is severe and renal function impaired, these may accumulate in extracellular fluid in the presence of total depletion. Infusion of potassium in this state would be hazardous and treatment is confined to the restoration of volume and the correction of concentration and acidosis. With dilution, restoration of renal flow, and insulin, rapid reversal of potassium shift may occur and the extracellular concentration of potassium may drop to dangerously low levels. At this stage infusion of potassium phosphate is indicated. Nabarro recently has infused magnesium salts in adults, but only very low concentrations would be safe in small children.

Pyloric stenosis exemplifies metabolic alkalosis from vomiting of chloride in excess of sodium. Despite the predominant position of chloride in stomach contents, sodium is lost in sufficient amounts to affect the ionic concentration of extracellular fluid and ultimately the volume. Bicarbonate takes the place of chloride with development of alkalosis and an increased pH value. Potassium moves from the cells and is replaced by sodium, the sodium shift-in being approximately two-thirds of the potassium shift-out of the cells, according to Cooke and his co-workers. This must disorganize cell activity and enzyme function. Potassium is lost from the body not only by renal excretion but also by secretion from distal tubules to a degree not revealed by serum biochemical investigations. Probably magnesium and phosphate are also lost. Tetany may occur.

It is noteworthy that when dehydration is severe, even in a state of severe alkalosis, acid urine may sometimes be excreted.

Other cases of intestinal obstruction can resemble pyloric stenosis or diarrhœa in electrolyte pattern, or can be free from distorting effect on acid-base equilibrium, depending on the site of obstruction.

Treatment.

The aim of treatment is to aid the restoration of homœostasis by providing the body with an arbitrarily but nevertheless carefully calculated infusion fluid which will fulfil the needs for restoration of volume, osmolarity, pH and pattern of intracellular and extracellular fluids, but at the same time place minimal strain on the regulators, namely, the kidneys and buffer systems, which in states of dehydration and pH change may have efficiency decidedly impaired. If one understands the underlying disordered physiology of a disease it is rarely necessary to rely on biochemical aids, although certainly they are useful when taken in conjunction with the overall picture.

For conditions of acidosis an alkaline solution is required, for alkalosis, an acid solution, and it must be remembered that the pH of extracellular and intracellular fluids is 7·35 to 7·45 and that neutral sodium chloride is actually an acid salt to the body.

Volume estimations in the absence of accurate serial weight charts from the commencement of the illness can be only arbitrarily assessed on clinical grounds for replacement purposes, but maintenance requirements are easier to estimate. There is always the danger of overhydrating small infants.

Concentration of infusion fluids for replacement purposes are also arbitrarily estimated on clinical grounds, and plasma electrolyte readings are of assistance for this. Maintenance is gauged on arbitrarily calculated daily abnormal losses together with the usual daily intake.

Intracellular electrolytes, particularly potassium, must be replaced when hydration has been restored.

In most cases of dehydration glucose is a necessary addition to infusion fluids because of its value (*a*) as a protein sparer, (*b*) as a means of reducing acidosis and hence the loss of base, (*c*) as a sodium sparer distinct from its reduction of acidosis, and finally (*d*) as a means of supplying calories.

FLUID AND ELECTROLYTE PROBLEMS IN THE NEWBORN.[1]

By Kate Campbell,
Melbourne.

The water content and the electrolyte pattern in the newborn differ from those of the older individual, as indeed does the chemical composition of the body as a whole, the baby being a chemically immature organism. According to McCance, working with fat-free tissue, the newborn body contains more sodium, chlorine, iron and copper than the adult, but less of all the other minerals.

Water.

Whereas water comprises 70% of the body weight of the adult, it comprises 80% of the body of the newborn. It is also differently disposed in that there is much more extracellular fluid and much less intracellular fluid than in the adult. The newborn has three times as much extracellular fluid, and about half the intracellular fluid of the adult. In both the plasma volume is the same, namely 5%. (See Figure I.) In adults 50% of the body weight is fluid inside

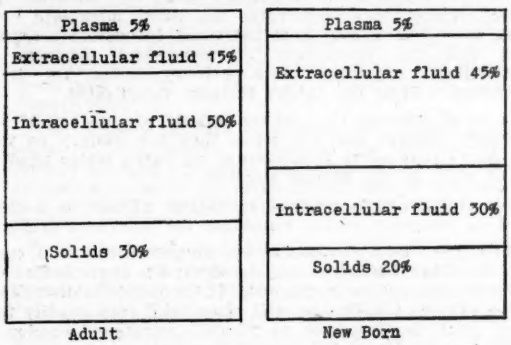


Figure I.

the cells, whereas in the newborn 50% is fluid outside the cells. As life advances there is a gradual dehydration, and a shift of water from the extracellular to the intracellular compartment.

In the premature infant there is still more water than in the full-time infant and a still greater percentage of extracellular fluid, while the cell mass is less than in the full-time child.

Electrolyte Pattern of the Infant.

The electrolyte pattern of the full-term infant shows differences from the adult pattern. The potassium content is higher. The chloride, sulphate and phosphate levels are higher and the bicarbonate and protein levels are lower, suggesting a compensated metabolic acidosis or a tendency thereto. In the cord serum, the potassium content is still higher than in the newborn serum and the sodium content is higher than the maternal level.

In the premature infant there is a higher concentration of base. The organic acid levels are higher and the bicarbonate level is still lower than in the full-time infant —that is, the tendency to acidosis present in all newborn is more marked in the premature infants. (See Table I.)

Acid Base Control.

The control of the acid-base equilibrium is relatively unstable and acidosis is more easily produced than in infants aged over two months.

[1] Part of a symposium on "Fluid and Electrolyte Problems", held at the annual meeting of the Australian Pædiatric Association, Canberra, April 10, 1953.

Renal Function in the Newborn.

It is held that the kidney does not produce its full number of glomeruli until the fœtus is thirty-six weeks old, whether this age is achieved inside or outside the uterus.

Renal function at birth in the full-time infant is immature and the immaturity is more marked in prematures of less than thirty-six weeks' gestation.

This immaturity is shown in the following ways:

1. The glomerular filtration rate is half that of older individuals so that the baby cannot get rid of sodium and chloride efficiently. If these ions are given in excess they will accumulate in the body with retention of fluid and the production of œdema.

2. Tubular function is ill-developed so that resorption of water is diminished. There is thus an inability to concentrate (or dilute) urine satisfactorily. The adult kidney can concentrate solutes to five times the plasma concentration, while the newborn can concentrate them only twice. The infant produces, therefore, hypotonic urine of low specific gravity, and has difficulty in conserving water.

3. Low phosphate content of urine makes it difficult to conserve base, therefore a tendency to acidosis is present. Normal function is reached in the second year.

Table I.

	Adult. (Milli-equivalents per Litre.)	Newborn. (Milli-equivalents per Litre.)	Cord. (Milli-equivalents per Litre.)
Sodium	140	140	142
Potassium	3·8–6	7·8	9·8
Chloride	103	107–110	—
Bicarbonate	27	20–22	—
Phosphate	25–45	5–8	—
Protein	16	14·8	—

Clinical Application.

Water Requirements.

It is difficult to find any authoritative work on the subject of water requirements and the amount required will vary with the following factors:

1. Length of gestation. The premature infant contains more fluid in its body than does the full-time infant. The post-mature infant with placental insufficiency is often clinically in a state of dehydration.

2. Early or late clamping of the cord. The average seven and a half pound baby, whose body blood volume is 10 ounces, with a late clamping of the cord will receive about three to three and a half ounces of blood from the placental circulation. This would be equivalent to giving a ten stone adult with a normal blood volume three and one-third pints of blood by transfusion. The infant accommodates the extra red cells in his vascular system and gets rid of the extra fluid into the extravascular extracellular fluid. (This adjustment starts in a quarter of an hour to three hours.) In the premature infant there is relatively more blood in the placenta than in the full-time infant; therefore if the cord is clamped late the premature infant gets a relatively larger vascular load.

3. Crying. This causes extra water loss in the exhaled air and by raising the basal metabolic rate increases the water requirement. "Crying for 1% of the time raises the basal metabolic rate 1%."

4. Sweating. Insensible perspiration is said to be *aqua destillata.* Sensible perspiration is hypotonic as compared with plasma.

5. Temperature at which the baby is maintained. A raised body temperature increases the basal metabolic rate and increases the water requirement. According to Mordhurst, quoted by Clement Smith, a lowered external temperature raises the basal metabolic rate, and therefore more water is required for excretion of end-products of metabolism.

6. The giving of glucose which reduces water loss in the urine.

7. Abnormal losses if vomiting or diarrhœa is present.

I have attempted to appraise clinically the water requirements in full-time infants whose crying was minimal and who slept most of the time. It would appear that in the first week a little over one ounce per pound of body weight per day seemed sufficient.

Gamble calculated the physiological fluid requirement of a 5% glucose solution (which is not quite the same as water requirement) in an eight pound baby and gave the figure as one and a quarter ounces per pound (80 cubic centimetres per kilogram).

Electrolyte Requirements.

In view of poor renal excretion one would expect the intake of electrolytes to be small. In breast milk there is a preponderance of the mineral cations over the mineral anions, which is unusual in foodstuffs. The proportion is approximately 2:1. Colostrum has slightly more of all the minerals than the later breast milk, and twice as much sodium, potassium and chlorine. Cow's milk has also a preponderance of cations over anions, but the ratio is lower —approximately 2:1½. The total electrolyte content is four times that of breast milk, the sodium being four times also that of breast milk. (See Table II, taken from Shohl's "Mineral Metabolism", 1939.)

TABLE II.

Electrolyte. (Ions.)	Breast Milk. (Milliequivalents per Litre.)	Cow's Milk. (Milliequivalents per Litre.)
Sodium	5·0	26·5
Potassium	12·2	39·5
Calcium	17·0	61·5
Magnesium	4·0	11·0
Total cations ..	38·2	138·5
Chlorine	10·0	32·8
Phosphorus ..	8·7	52·0
Sulphur	2·2	19·4
Total anions ..	20·9	104·0
Grand total ..	59·1	242·2

Attention has been drawn lately to the fact that newborn infants fed on artificial feedings containing high amounts of sodium (such as whole cow's milk, or strong cow's milk mixtures, or carbohydrates which contain sodium chloride) showed œdema and large gains in weight. The œdema disappeared and the infants lost weight on iso-caloric feedings of breast milk. It was suggested that this œdema might play a role in the ætiology of retrolental fibroplasia.

It has also been pointed out that the giving of whole cow's milk increases the solute load on the kidney. This increases the obligatory urinary water requirements and reduces the margin of safety if any dehydrating condition should occur.

It would appear, therefore, that in planning artificial feeding we must take cognizance of electrolytes.

The parenteral administration of normal saline also produces œdema. It is therefore our practice to use N/5 saline with 4% glucose for young infants.

Calcium.

Serum calcium content, which at birth is 1·0 milligramme per 100 mils above the maternal level, falls in the first forty-eight hours (11·27 to 9·93 milligrammes per 100 mils). The serum phosphorus content rises after birth. Bakwin suggests these changes are due to a physiological relative hypoparathyroidism. If the infant is given a feeding rich in phosphorus and with a low calcium:phosphorus ratio—for example, cow's milk feeding—and the kidney excretes phosphorus poorly, it will accumulate with a consequent fall in the calcium content and tetany may result. Tetany occurs more commonly in the artificially-fed than in the breast-fed.

Treatment suggested is 2% solution calcium gluconate given intravenously: 0·3 to 1·0 gramme injected slowly. Intramuscular treatment is not advised, but I have given four minims of 10% calcium gluconate solution per pound body weight without trouble.

Dehydration.

Dehydration may be produced (*a*) by simple water loss without electrolyte loss (for example, dehydration fever of the newborn); (*b*) by loss of water and electrolytes, as in vomiting and/or diarrhœa (this is considered by other speakers); (*c*) by delayed feeding.

Dehydration Fever.

These infants cry a great deal, thereby losing water vapour in excess in the exhaled air, without a loss of electrolytes. As a result, the extracellular fluid becomes hypertonic and water passes from the cells to the extracellular fluid, thus producing a cellular dehydration (hypertonic dehydration). The restlessness and agitation cause sensible perspiration which in the newborn has a low electrolyte content, and so further water loss ensues. The raised temperature accelerates the basal metabolic rate, with more water loss from increased insensible perspiration and the production of more end products of metabolism, requiring extra urinary water for their excretion. Thus the infant becomes dehydrated.

It is of interest that where infants are nursed at the mother's bedside and fed when they are hungry so that crying is reduced to a minimum, no extra water need be given even in the hottest weather.

The infant with cerebral irritation who cries a great deal is always a likely candidate for dehydration fever.

The free administration of 5% glucose (or water) cures the condition, though it may be thirty-six hours before the normal temperature is reached. If the infant is disinclined for a glucose solution he will often take very readily to a very weak feeding such as Nestlé's sweetened condensed milk one in 24 (6 Calories per ounce).

Occasionally the baby may refuse sufficient fluid. He may then be given 5% glucose in *aqua destillata* intravenously.

Delayed Feeding.

Two reasons have been given for delayed feeding, which has been practised mainly with premature infants. Originally it was done to "dry the babies out". Actually we cannot rid the babies of water without at the same time ridding them of electrolytes. The other reason, and the more rational one, is to give the babies rest.

The delayed feeding may be done in three ways: (*a*) by giving water alone (fasting the baby); (*b*) by giving 5% glucose solution (partial fasting); (*c*) by giving nothing at all (fasting and "thirsting" the baby).

The following are the results in adults. Infants, because of poor renal function, conserve water less well. Also the intake and outgo of water are greater in comparison with the total body water than in adults, so that they become dehydrated more rapidly.

Giving Water Alone.—Giving water alone means the withholding of electrolytes and food (hypotonic dehydration). The procedure has the following effects: (*a*) By starvation it increases organic acids and aggravates acidosis. (*b*) It causes consumption of body protein. Each gramme of protein leaving the cells causes loss of 3·0 mils of intracellular water which takes with it the intracellular electrolytes. This loss of tissue water cannot be prevented by giving water. (*c*) Loss of extracellular electrolytes occurs in urine so that extracellular fluid becomes hypotonic and loses fluid to the tissues. Therefore there is a decrease in extracellular fluid, a decrease in serum electrolytes and a decrease in plasma

volume. (*d*) Potassium may leave the cells to replace sodium, and potassium is then excreted, with resulting potassium depletion (ileus, hypotonia *et cetera*).

Giving 5% Glucose Solution.—The giving of 5% glucose solution (*a*) prevents acidosis, (*b*) reduces protein loss to half, (*c*) reduces sodium loss to half (Gamble); it therefore reduces water loss and reduces the risk of potassium depletion.

Giving Nothing At All.—The effects of giving nothing—that is, the withholding of water, electrolytes and food—may be enumerated as followed: (*a*) acidosis; (*b*) consumption of body protein; (*c*) increased loss of tissue fluid without further electrolyte loss giving increased concentration of intracellular electrolytes; (*d*) increased loss of extracellular fluid, at first isotonic, later with more loss of water than of electrolytes—therefore a rise in the concentration of serum electrolytes; (*e*) dehydration; (*f*) hæmoconcentration as shown by hæmatocrit, causing increased concentration of bilirubin (and allied substances); (*g*) hypoglycæmia. The blood glucose level is low in the newborn and lower still in premature infants. Glycogen is exhausted two hours after birth in full-time infants and earlier in premature infants, whose livers have less glycogen than full-time infants.

In Hansen's series of infants given nothing for three days, the weight loss was 13·5%. Those given water lost 8·3%. In those given nothing there was a rise in the serum sodium content (12 milliequivalents per litre), chloride (7 milliequivalents per litre) and urea (9·0 milligrammes per 100 mils), whereas in those given water there was a fall in all these substances (4, 3·5 and 3 respectively).

The urinary outputs of sodium and chloride were the same in both groups. Although the serum electrolytes showed marked increases, and it is certain that there was an increase also in the intracellular electrolytes, the infant had no rise in temperature and no untoward signs were noticed.

Clinically it is my impression that the premature infants begin to look dehydrated after forty-eight hours, and I like to start fluid administration then.

If a premature infant shows marked early jaundice I think it is unwise to delay feeding too long. If at the end of forty-eight hours the infant is not able to take reasonable amounts of fluid, I think it advisable to start a subcutaneous drip with hyaluronidase, in order to overcome the hæmoconcentration and to increase the amount of urine. The combination of a high serum bilirubin content and an unnaturally low blood glucose level seems to me to constitute a threat to the brain tissue and to be a possible cause of "premature kernicterus".

The drip should not be given too rapidly, as it will then distend the tissues and prevent good absorption. A rate of drip giving about two ounces per pound per day is generally well tolerated and a suitable solution is N/5 saline with 4% glucose, or 5% glucose in *aqua destillata*.

Œdema.

Œdema in the newborn is a very difficult subject. It has been pointed out that the prevention of œdema requires an intact circulatory-renal-hormonal mechanism.

Very premature infants show marked pitting œdema, often as early as one hour after birth. Full-time infants show a "smudginess" of the face and some swelling of the eyelids from about the second day or earlier which is due to œdema. The readjustment of the circulation to the load of placental blood probably plays some part in this.

I do not think that this occurs in the post-mature infants who show evidence of placental insufficiency and dehydration.

Rough Treatment of Persistent Vomiting Without Benefit of Laboratory.

Most of these patients will have been suffering from intestinal obstruction. A rough clinical basis is as follows. Give intravenous drip of N/5 saline with 4% glucose at the basal rate of one and a half ounces per pound of body weight per day, with sufficient added to make up for vomitus or amount aspirated. After forty-eight hours give serum in amount equal to half an ounce per pound of the baby's body weight. At seventy-two to ninety-six hours 0·25-0·5 gramme of potassium chloride is added to 500 mils of solution given intravenously. One the fifth or sixth day blood in amount equal to half an ounce per pound of body weight is given.

THE PATHOLOGICAL PHYSIOLOGY, ASSESSMENT AND MANAGEMENT OF "DEHYDRATION" IN ENTERO-COLITIS.[1]

By HOWARD WILLIAMS,
Melbourne.

"DEHYDRATION" is by far the most important factor causing morbidity and death in infants with entero-colitis. Understanding of the disordered water and electrolyte metabolism leading to dehydration, and assessment of this disordered state are essential for correct management. It is not enough to give an intravenous infusion of sodium chloride with water until the patient looks better clinically, for while this may suffice in some instances, in many it will not and death may follow. Clinical practice must keep pace with our knowledge of the deranged physiology if more lives are to be saved and the morbidity reduced.

Pathological Physiology.

The infant with diarrhœa loses water, sodium, potassium, chloride and bicarbonate ions in the stools in variable and unpredictable amounts, and these are often not adequately replaced owing to vomiting and difficulty in feeding. Depending on the extent and severity of the losses of water and electrolytes, a series of changes occurs. Predominant water loss results in the reduction of extracellular water, and a shift of water from the cells occurs with consequent hyperosmolarity of the extracellular and intracellular fluid. This shift favours the maintenance of circulating blood volume, although in the acute severe stage of dehydration the reduction of the extracellular fluid and blood volumes may be sufficiently great to cause circulatory embarrassment and later failure. Circulatory failure, together with an acute reduction in cell water, will lead to failure of cell metabolism and eventually death. Acidosis frequently occurs as a larger quantity of sodium than chloride is lost, and ketoacids accumulate in the body. It must be emphasized that in the early acute stage the important losses are water, sodium and chloride, and these must be restored rapidly to relieve the circulatory embarrassment and to restore cell function. Should the diarrhœa be of short duration and the infant able to take adequate feedings orally, dehydration does not develop.

With persistent diarrhœa the body may gradually be seriously depleted of electrolytes, the two most important being potassium and sodium. When the cells have lost considerable quantities of potassium, serious derangement of cell function occurs and is usually manifested clinically by gross hypotonia, muscle weakness and cardiac failure. Sodium depletion, if severe, leads to increasing dehydration and circulatory failure. The patient with persistent diarrhœa is usually supplied with adequate quantities of water, but not infrequently with insufficient electrolytes, especially potassium. The chief clinical problem in a patient who is losing ground with continued severe diarrhœa is to assess whether chronic potassium or sodium depletion or both are present.

Assessment.

In clinical practice it is essential to attempt to evaluate both the type and degree of water and electrolyte disorder.

[1] Part of a symposium on "Fluid and Electrolyte Problems", held at the annual meeting of the Australian Pædiatric Association, Canberra, April 10, 1953.

This assessment can be reasonably accomplished mainly on clinical evidence; biochemical tests help at certain stages, but are not essential in the early acute phase. Discussion will be clarified if cases are divided into the early severe acute group and the group of some days or weeks' duration.

In the early acute severe type information about the duration and degree of diarrhœa together with the oral fluid intake will indicate the probable degree of water depletion. It has been shown that approximately 300 mils of water are lost daily in diarrhœa of moderate severity and up to 500 mils in the more extreme case. Weight loss, if figures are available, is also a good guide. If the infant is sunken-eyed, listless, with cold extremities, slight peripheral cyanosis, a poor capillary circulation and a rapid weak pulse, his water loss is extreme, and unless it is rapidly replaced death will supervene. In severe dehydration one tenth and even more of the total body weight may be lost as water. Every gradation is seen from this extremity to that manifested by the infant who is restless, flushed, irritable and feverish from mild water loss. The general clinical appearance of the infant, together with the state of his circulation, affords the most reliable guide of the degree of dehydration. Intravenous medication is imperative for saving life if the circulation is severely embarrassed. Oral feeding usually suffices in the mild to moderate case. In the early acute group serum electrolyte levels are not essential either to determine the type of disorder or to decide what treatment to give.

The patient with persistent diarrhœa, who is difficult to feed and who has had intravenous therapy either continuously or intermittently, can often become seriously depleted of certain electrolytes, the most important ones being potassium and sodium. Extensive disturbances of electrolyte patterns may slowly develop with few clinical signs, and it is only when the compensating mechanisms of the body fail that signs and symptoms develop in an alarming and rapid manner. Assessment prior to collapse may be very difficult, but it is usually possible to be aware that serious depletion is developing by weighing the patient and recording the quantities of water, sodium and potassium taken daily. If the patient is steadily losing weight and the intake of either sodium or potassium or both is very low over some days, the body is almost certainly depleted in the ions of either, or both. Serum electrolyte estimations may be of considerable help in diagnosis; a low sodium concentration indicates sodium depletion, a low potassium concentration usually indicates potassium depletion, but this is by no means excluded by a normal figure.

Treatment.

In the acute severe case with circulatory failure the patient requires urgent intravenous infusion to restore his circulating blood volume, extracellular water and electrolytes, and to correct his acidosis. As water has been lost in excess of electrolytes and sodium in greater proportion than chloride, the fluid given should be half isotonic sodium chloride with 30 to 40 mils of molar sodium lactate and 25 to 50 grammes of glucose to the litre. An amount equal to one tenth of the body weight of this solution is given intravenously, one third of the total amount rapidly in half to one hour and the remainder over three to six hours. With this treatment almost all infants rapidly improve and the circulation is restored. In the rare case in which circulatory failure is not relieved, a transfusion of serum, 60 mils per kilogram of body weight, should be given. After this initial treatment it is recommended that infusion be continued with one fifth or one fourth isotonic sodium chloride containing 5% glucose at a rate of 180 mils per kilogram of body weight over the following twenty-four hours.

The key to treatment at this stage is to make sure that adequate quantities of water, sodium, potassium, phosphate and glucose are given daily. The amounts given are estimated on a basis of average daily losses in diarrhœa and average daily requirements, and the following practice is based on these figures. After the initial period of twenty-four to thirty-six hours' fasting, glucose water to which is added an electrolyte mixture each 30 mils (one ounce) of which contains 14 milliequivalents of sodium, 10 milliequivalents of chloride, 8 of potassium, and 3 of phosphate is given. (The following is the formula for preparation: sodium chloride 10 grammes, sodium citrate 36 grammes, potassium chloride 16 grammes, potassium acid phosphate 15 grammes and water 40 ounces.) According to the daily intake of sodium required the corresponding number of ounces of electrolyte mixture are added to the day's fluid intake. A baby weighing five kilograms requires approximately three to four ounces daily, a baby weighing 10 kilograms four to six ounces daily. Intravenous therapy is gradually discontinued as the infant takes his full water requirements by mouth. Usually after one, or in the more severe cases two, days of the giving of water-glucose-electrolyte mixture by mouth, dilute milk feedings can be given, but it is wise to continue adding some electrolyte mixture to the milk until full-strength milk is resumed and the diarrhœa greatly reduced.

If the infant is only mildly or moderately dehydrated and does not require intravenous therapy, a trial of oral feeding is made, 5% glucose water being used to which have been added corresponding amounts of electrolyte mixture. Water requirements for the day are estimated on the basis of 150 mils per kilogram body weight *plus* one twentieth body weight to allow for dehydration. Three to six ounces of electrolyte mixture (according to the size of the infant) are added to the day's water-glucose requirements, and the mixture is given either by slow, continuous intragastric drip or in evenly divided doses at hourly intervals. If these quantities are retained during the first day, half-strength skimmed milk with electrolyte mixture is given on the second day, when the infant is better fed every two or three hours. As the diarrhœa lessens, the strength of the milk is increased and the electrolyte mixture gradually discontinued. Should the infant vomit and not be able to take his fluid requirements orally, intravenous therapy will be necessary. The above régime has been well tested for over two years at the Children's Hospital, Melbourne, and has been found to be both safe and efficacious, and has prevented the development of chronic electrolyte depletion.

The severely ill infant with persistent diarrhœa who is steadily losing weight presents a difficult problem. Chronic sodium or potassium depletion must be considered as a probable cause of his failure to progress. If the daily intake of either sodium or potassium is constantly low, then depletion is very probable, and estimation of the level of these electrolytes in the serum is necessary. A low sodium level indicates sodium depletion, and low potassium level potassium depletion, but it is well to note that a normal serum potassium concentration does not exclude severe depletion of cell potassium. As a general rule sodium depletion is usually due to failure to add extra sodium to dilute milk feedings when diarrhœa persists, and potassium depletion is usually due to long-continued intravenous therapy without the administration of potassium. Since the institution of the type of treatment outlined for the acute case no patient has suffered with either chronic sodium or potassium depletion in the last two years in the Children's Hospital, whereas previously it was not infrequent. If sodium depletion is present, it is advisable to give one tenth body weight of isotonic sodium chloride, over four to six hours, and to continue with maintenance therapy as previously outlined. It is necessary to treat severe potassium depletion with 3 milliequivalents of potassium per kilogram of body weight given over twenty-four hours. Intravenous therapy is usually necessary early, but potassium should be given orally as soon as possible. For intravenous use a solution of 2·0 grammes of potassium chloride and 2·0 grammes of potassium phosphate (KH_2PO_4) in one litre of one quarter isotonic or one fifth isotonic sodium chloride containing 5% glucose can be used and an amount given to supply 3 milliequivalents of potassium per kilogram of body weight per day. One litre of this solution contains approximately 40 milliequivalents per litre of potassium. When the patient can take fluid orally, additional potassium can be added to the electrolyte mixture described previously.

The best treatment of chronic or subacute electrolyte depletion in infective diarrhœa is prevention. If the clinician keeps good records and makes sure that his patient has an adequate intake of water, sodium, potassium, chloride and phosphate received either orally or intravenously or by both routes, then it is most improbable that these disorders will develop.

All seriously ill or dehydrated infants should be treated in hospital, but in the mild case the patient is treated best in the home. In home treatment it is essential to have a simple plan with clear instructions. The infant should be starved of all food, and given only water and glucose for a period of approximately twenty-four hours. The quantity of water and glucose to be given over this period is estimated on the basis of a daily requirement of 150 mils per kilogram of body weight, *plus* an additional amount to make up for loss in diarrhœa 200 to 300 mils. Water containing 5% glucose is best given with orange or lemon juice or other fruit juice and with 5·0 grammes of sodium chloride added to each litre. The estimated amount is given over twenty-four hours in evenly divided quantities at half to one hourly intervals, due allowance being made for periods of sleep.

FLUID AND ELECTROLYTE PROBLEMS IN INFANTS AND CHILDREN: INTESTINAL OBSTRUCTION.[1]

By E. S. STUCKEY,
Sydney.

I PROPOSE to discuss, from the practical point of view, the management of a patient with complete intestinal obstruction. The patient in mind will be one with jejunal or upper ileal obstruction who is about to undergo surgical operation or has already done so. I consider that the patient with paralytic ileus and peritonitis, whether pre-operative or post-operative, comes within the scope of this discussion. Only passing references will be made to purely gastric obstruction (for example, pyloric stenosis) and purely colonic or lower ileal obstruction (for example, intussusception).

THE PROBLEMS.

All such patients are suffering from a "mixed" form of dehydration due to at least two factors: (*a*) abnormal losses of intestinal secretions by vomiting, aspiration, or fistula; (*b*) failure to take or retain normal fluids for maintenance purposes. These patients therefore require a period of total intravenous alimentation until both these factors have been overcome.

They may require, in addition, special fluids to prevent or to overcome surgical shock due to loss of circulating red blood cells and/or plasma proteins. The shock may be due to either (*a*) the disease process (for example, strangulation); (*b*) the operation necessary to cure them (for example, bowel resection).

Dehydration may be a problem both before and after operation. Operation must be considered as an important "incident" in the course of treatment and not as the sole or main remedial measure. In all except mild degrees of dehydration special pre-operative "resuscitation" is required; otherwise there is likely to be a high mortality. The operation itself may be of such a nature as to produce or to aggravate surgical shock, and provision must be made for this at the time. In addition, operation further upsets the fluid and electrolyte balance, being followed by a period of sodium retention and of increased protein breakdown. This is probably a product of adrenal "stress".

Renal function is depressed in severe dehydration because there is not enough water to spare for the kidneys to excrete the accumulating waste products. An infant cannot concentrate these waste products in his urine as well as an older child and therefore withstands dehydration worse than an older child. It is essential to supply sufficient fluid rapidly in such cases in order to allow the kidneys to regain their ability to discriminate between substances to be retained and those to be excreted. It is assumed that the patient had healthy kidneys prior to the onset of the disease. It is further assumed that once sufficient water and electrolytes have been supplied, the kidneys will be able to overcome moderate degrees of acidosis or alkalosis without provision of special fluids for these conditions, except in very severe cases.

Age and Weight.

It is easy to lay down rules for an average adult. When discussing treatment in infants and children it is necessary to use calculations based on average weight at different ages. In small infants this is not sufficiently accurate and it is necessary to use known weight prior to onset of dehydration, or estimated weight calculated from a knowledge of age, birth weight, dehydrated weight, and clinical degree of dehydration.

In practice it is convenient to save calculation by working from tables which show for various weights and ages: (*a*) the basic daily maintenance needs in health; (*b*) replacement needs for moderate dehydration; and (*c*) additional needs of saline or serum for severe dehydration or for surgical shock.

Laboratory Aids.

All laboratory tests involve delay and they should be used more as measures of the progress of treatment than as a guide to treatment. Clinical judgement is essential. Serum electrolyte determinations, if measured by rapid methods, are a help, especially sodium and chloride levels. Serum potassium levels are of limited value, as they are not a measure of the extent of depletion of cellular fluids or of the quantity needed to restore normality.

Hæmoconcentration measurements are useful in assessing progress of treatment, especially when surgical shock is a factor.

No reliance can be placed on one isolated measurement of urinary chlorides, and a report on a twenty-four hour specimen is useless as it is at least twenty-four hours too late. Nevertheless it is considered that ward tests of urinary chlorides on all available samples are a help in assessing progress. Chloride test papers are very simple to use as a qualitative test.

Method of Recording Fluid Balance.

Considerable thought has been given at the Royal Alexandra Hospital for Children to the problem. We have devised two new charts which we hope will make it possible to keep more accurate records in future, and will simplify the method of assessing "today's needs". (*a*) One is a daily intravenous fluid record which is filled in in detail by the resident medical officer and on which the nursing staff enters each stage as completed. (*b*) The other is a daily "intravenous fluid balance" chart on which the resident medical officer summarizes the deficit and estimates "today's fluid requirements" before writing out the daily intravenous fluid record.

In assessing the rate of drip the following formula is useful: three drops per minute = 10 mils per hour
three drops per minute = 250 mils per 24 hours.

DETAILS OF TREATMENT.

A. Resuscitation (First Two to Four Hours).

On the patient's admission to hospital a careful clinical estimate of the degree of dehydration and of the presence of absence of surgical shock should be made. If the estimate is one of mild to moderate dehydration, resuscitation is not needed and treatment begins as outlined in the next section (B).

Resuscitation treatment must be considered under two headings:

[1] Part of a symposium on "Fluid and Electrolyte Problems", held at the annual meeting of the Australian Pædiatric Association, Canberra, April 10, 1953.

1. Surgical shock may be part of the clinical picture on the patient's admission to hospital (for example, in intestinal strangulation) or it may arise *de novo*, even in the moderate case, at or after operation. Whenever surgical shock is considered to be present, its treatment should take precedence over other measures, and shock treatment should be given, as rapidly as possible, and other calculated fluid needs should be given later.

The appropriate fluid is blood or serum in sufficient quantity to replace estimated plasma loss. From 25% to 30% of blood volume is a suitable "unit volume" to administer, and a convenient formula in infants is 2% of body weight (10 mils per pound of weight). On the child's admission to hospital it is reasonable to commence with some replacement fluid while obtaining the blood or serum.

2. In severe dehydration abnormal fluid loss commonly amounts to 10% to 15% body weight, and has occurred largely from the interstitial fluids and partly from the cells. A large proportion of the abnormal fluid loss should be restored within the first two to four hours of treatment, even in the absence of surgical shock. Abnormal fluid loss from any source, whether vomitus, gastric or intestinal aspiration, ileostomy, fistula or even excessive sweating may be assumed to cause the loss of at least 0·5 gramme of sodium chloride per 100 mils (86 milliequivalents per litre).

It is recommended that in severe dehydration the preliminary "resuscitation treatment"[1] to be given in the first two to four hours should be: (*a*) Serum to a least 2% of body weight. (*b*) A similar quantity of N/2 saline-glucose solution (normal saline mixed with an equal quantity of 5% dextrose in distilled water). This solution contains 0·45 gramme of sodium chloride per 100 mils (77 milliequivalents per litre). This is in addition to true "shock therapy". In a "desperate" case even more would be given. Operation on patients requiring initial resuscitation should be postponed for two to four hours until this treatment has been given. If resuscitation has been adequate there should be a copious flow of urine.

B. Correction of Moderate Dehydration (First Twenty-four Hours).

The moderately dehydrated patient, or the severely dehydrated patient after his initial period of resuscitation should be estimated as requiring in the first twenty-four hours:

(i) Replacement fluids equal to approximately 6% body weight (30 mils per pound of weight).

(ii) Maintenance fluids for twenty-four hours. Quantities are obtained from tables and the total amount should be given as follows: (*a*) one half in the first six hours, (*b*) one quarter in the next six hours, (*c*) one quarter in the remaining twelve hours.

Replacement fluid should be given first and maintenance fluids later. The initial period of treatment including resuscitation should be one of approximately twenty-four hours, irrespective of the hour of the commencement of treatment. The patient's clinical progress should be reassessed at least every twelve hours, and the estimate of treatment needed may have to be revised either up or down. It should be the aim to have the initial deficit practically overcome at the end of twenty-four hours.

Replacement Fluids.

In my opinion replacement fluids should contain potassium as well as sodium chloride, except perhaps in the initial period of resuscitation. A suitable fluid contains: 4·5 grammes of sodium chloride per litre (77 milliequivalents), 0·5 gramme of potassium chloride per litre (7 milliequivalents), made isotonic with dextrose or fructose. This fluid should be perfectly safe, as in theory potassium does no harm unless the plasma potassium level rises above 10 milliequivalents per litre, and William Steiger asserts that while there is a remaining deficit of potassium in the cells it will be rapidly removed from the plasma. An alternative fluid for those who consider the administration of potassium unwise at this stage is N/2 saline-glucose which contains 4·5 grammes of sodium chloride per litre.

Maintenance Fluids.

Infants need fluids for maintenance purposes to the extent of 15% body weight daily (70 mils per pound weight). During the first week of life less is needed. A useful calculation during this period is 10 mils per pound weight on the first day, 20 mils on the second day, and so on. Older children require less than 70 mils per pound weight. Appropriate amounts vary from less than 500 mils daily to 2500 at the age of fifteen years. Maintenance fluids must contain appropriate quantities of electrolytes, in particular sodium and chlorine ions. Maintenance needs for salt are small. Katherine Dodd quotes: under one month, 0·25 to 0·5 gramme (presumably given by mouth); up to six months, 0·5 to 1·0 gramme; aged two years, 1·5 to 2·0 grammes; older children, 2·0 to 4·0 grammes.

In the past we have given maintenance fluids as follows: sufficient N/5 saline with 4% glucose solution is given to provide the salt needs, and the balance is made up as 5% glucose in distilled water. A more practical method, probably, is to have appropriate maintenance fluids for different ages.

I believe that maintenance fluids should contain potassium as well as sodium chloride, and offer the following suggestions:

1. Basic maintenance fluid containing: sodium chloride, 0·4 gramme per litre (7 milliequivalents), potassium chloride, 0·2 gramme per litre (2·8 milliequivalents) made isotonic with dextrose and suitable as the sole maintenance fluid for infants up to six months of age (one litre).

2. Supplementary maintenance fluid five times as strong as the basic maintenance fluid and containing sodium chloride, 2·0 grammes per litre (35 milliequivalents) and potassium chloride, 1·0 gramme per litre (14 milliequivalents) made isotonic with dextrose. This is given to older infants and children in addition to one litre of the basic maintenance fluid as follows: 100 mils per year of age. For example, at four years of age, give one litre of basic maintenance fluid *plus* 400 mils of supplementary maintenance fluid.[1]

C. Second Day.

The second "day" commences at the end of the first twenty-four to twenty-seven hour period of treatment. The hour of commencement is variable, but it is recommended that the second "day" should end at a fixed hour (for example, 9.30 a.m. next morning) and that thereafter each day's treatment should begin and end at 9.30 a.m. For this reason all measurements of fluid given and of abnormal fluid losses must be made by the nursing staff at, say, 9 a.m. daily. The resident medical officer can then check the fluid given "yesterday" from the daily intravenous fluid record, can complete "yesterday's" "intravenous fluid balance" chart and carry forward any remaining deficit to "today's" fluid balance chart and then fill in in detail "today's" intravenous fluid record. The second "day's" fluid needs will consist of (*a*) residual deficit not yet overcome, (*b*) maintenance needs for *X* hours to 9.30 a.m.

There will usually be a residual deficit to make up, owing to two factors: (*a*) Failure to achieve the programme laid down; (*b*) abnormal fluid losses—for example, gastric suction during the first day of treatment.

The fluids used are exactly as before—namely, replacement fluid and maintenance fluid according to age.

Of course, clinical or laboratory evidence might lead to the impression that the patient has been either overtreated or undertreated and the programme may be modified accordingly, a suitable remark being made on the fluid balance chart.

[1] Dr. Stuckey illustrated his paper by charts and tables which have not been reproduced here.

[1] It must be stressed that sufficient maintenance fluid must be given intravenously daily, in addition to that required to meet replacement needs, until the patient is taking and retaining fluids by mouth.

D. Third and Subsequent Days.

The initial deficit should by now be completely overcome, and the only replacement fluid needed should be the quantity vomited or aspirated "yesterday". This must continue to be measured and entered on the fluid balance chart daily until oral feeding is resumed. At this stage consideration should be given to: (*a*) protein replacement, (*b*) vitamin needs, (*c*) the provision of blood to remedy the anæmia

Maintenance Fluids with Protein or Aminoacids.

Parenamine can be added to maintenance fluids to the extent of 1·0 gramme per pound weight (daily) of protein for infants. Parenamine to be given intravenously in 5% solution contains 1·0 gramme of protein equivalent in each 20 mils. It contains negligible quantities of electrolytes. A method of obtaining suitable maintenance fluids with parenamine is as follows. Parenamine maintenance fluid (basic) would contain 300 mils per litre of 5% parenamine in place of part of the glucose in the basal maintenance fluid and would be used exactly as basic maintenance fluid is used. After the age of six months one litre of this would be used, and in addition a supplementary parenamine solution containing 500 mils or more of 5% parenamine per litre in place of part of the glucose in the supplementary maintenance fluid. This would be given as supplementary maintenance fluid is given and would provide a decreasing scale of protein replacement in older children.

Vitamins.

Both mixed vitamin *B* concentrate and vitamin *C* should be given by injection daily or every two days. Crude liver extract is also of value.

Blood.

If hæmoglobin tests at this stage reveal anæmia a (further) transfusion of a unit volume (or less) of blood should be given, in addition to maintenance fluids, to correct the anæmia.

Mouth Feeding.

As soon as peristalsis is present and flatus is being passed it is safe to discontinue gastric aspiration, and soon thereafter small quantities of fluid may be given by mouth, correspondingly less maintenance fluid being given intravenously. When sufficient fluid, preferably milk, is being retained by mouth, intravenous fluid therapy can be discontinued, usually on the fourth or fifth day.

THE MINOR EPILEPSIES.[1]

By D. G. HAMILTON,
Sydney.

PETIT MAL is a vagabond term which to most people means any mild or brief seizure. This is a pity, for minor seizures may be of some half-dozen types that differ in clinical form, electroencephalographic pattern, drug response and outlook. They include true *petit mal*, myoclonic and akinetic epilepsy, which are related in that they have a spike-wave pattern in the electroencephalogram, but also minor motor, mild psychomotor and autonomic fits. I would prefer the term "*petit mal*" to be restricted to what Lennox, borrowing from the French, would call "*petite absence*", or what some people call classical *petit mal*. It is one of the more common of the minor seizures. It is a brief oblivion, lasting from some five to thirty seconds. The patient stops and appears in a trance, unconscious of surroundings and impervious to interruption. There may be fluttering of the eyelids, gentle rapid rhythmic movements of the hands or head or slight staggering. Recovery is rapid; perhaps a shake of the head and a few blinks and the patient resumes his interrupted activity. There is no aura, no violent jerking, no cyanosis and no post-convulsive confusion or sleep. These turns may occur once each few days or many times a day. Often they may be produced by overbreathing. They seldom happen while the patient is concentrating on mental or physical activity, but rather while the mind is idle. The resting electroencephalogram is often normal, but on overbreathing or during a fit a three cycles per second spike and wave rhythm, precise and machine-like in its regularity, appears. Most often these fits are idiopathic and the only type of seizure occurring. Some patients, however, have occasional major fits as well, and in some they are the sequel of brain damage, though more often one finds here a *petit mal* variant.

True *petit mal* responds best to the diones, "Tridione", "Malidone" or "Paradione". If one fails, another may succeed. About half the patients have their fits quickly and completely controlled. Some show partial response and some none at all. It is often said that the barbiturates make *petit mal* worse. This is not always so. Often phenobarbital or "Prominal" will reduce the frequency of fits, and in a few paients control them completely. If diones fail, barbiturates are certainly worthy of trial. Indeed, I often reverse the process, and try barbiturates first, for they are safe and relatively cheap, and do not require the close watch on blood and urine that is essential when the diones are used. "Dilantin" may make these fits worse.

These fits, though occurring frequently, do not cause mental deterioration. Often they cease at puberty or during adolescence, and the outlook generally is good.

The second of the minor epilepsies is the *petit mal* variant. The form of the fit is identical with that of true *petit mal*, but the form of the electroencephalogram is different. Spikes and waves occur, but they are slower and less regular than in true *petit mal*. They may occur in short runs or as isolated entities scattered throughout a record that tends to be abnormal and irregular in its basic rhythm. And much more often there are other clinical features; other types of fit may be prominent; there may be evidence of brain damage. The response to treatment, uncertain enough in true *petit mal*, is still more capricious in its variant. A greater proportion of the fits defy drug control. Nor can one be so confident that diones are likely to be the most effective drugs. Often enough they are, but if they fail, any of the whole range of anticonvulsants—barbiturates, "Dilantin", "Mesantoin", "Mysoline", "Milontin", bromides, "Dexedrine"—is worthy of trial. Nor can one be so optimistic about the ultimate outlook of the children who have fits of various sorts, among them this *petit mal* variant, who too often have brain damage, and whose relief from drugs may be disappointing.

It will be seen that the most important distinction between true *petit mal* and its variant is in the rate of the spike and wave discharges, the three cycles per second rate of *petit mal* and the slower rate of its variant. Lennox (1950) has studied this distinction in a large group of patients. With either type of rhythm there may be *petit mal*, myoclonic or akinetic fits. With either type there may occur other fits, *grand mal* or psychomotor, whose characteristic rhythm is not a spike-wave one. But Lennox found important differences, namely:

1. The background rhythm was grossly abnormal in twice as many (31%) of the slow group as of the three cycles per second group (15%).

2. Almost half the slow group had no *petit mal*, myoclonic or akinetic seizures, but three-fourths of them had convulsive or psychomotor fits (with or without *petit mal*).

3. There was clinical evidence of brain damage in 75% of the slow group, but in only 4·5% of the three cycles per second group.

4. There was a genetic background of epilepsy in a greater proportion of the fast group.

The myclonic jerk is the next of the minor epilepsies. It is a sudden single convulsive jerk of one or several muscle groups with no apparent unconsciousness. The

[1] Read at the annual meeting of the Australian Pædiatric Association, Canberra, April 10 to 12, 1953.

forms I have seen most commonly are the sudden outflinging of both arms and flexing of the body in mentally defective infants, and the sudden vigorous flexion of the whole body perhaps with a throwing up of the arms in older children. However, it may be a sudden jerk of the head or a limb. This may be the only fit occurring in an otherwise normal child, or it may be but one of several types of fit in a child who is likely to have brain damage. Usually the electroencephalogram shows the same sort of tracing as in the *petit mal* variant, and there is the same unpredictable response to treatment. The spike-wave rhythm may be the three cycles per second rhythm of *petit mal*, but then the jerks are localized and not massive, and evidence of brain damage is rarely present.

The akinetic fit is a sudden loss of postural tone of the whole or part of the body, and results in such fits as head-nodding or drop seizures. The head suddenly falls forward onto the chest and is immediately lifted, or the patient suddenly falls to the floor, but rises again immediately and clear-headed. Again the electroencephalogram is likely to show the fast or slow spike-wave pattern.

There remain some minor fits that are not characteristically associated with a spike-wave pattern in the electroencephalogram.

The minor motor fit is common. It is a very mild or incompletely developed major fit. It may be very brief, often of no greater duration than *petit mal*. But its components are those of a major fit. There may be vigorous clonus, or cyanosis, or an aura, or unconsciousness that is deeper or longer than in *petit mal*, or post-convulsive confusion or sleep. These are not features of *petit mal*. They may not all be present. Perhaps only one of them is there to reveal the true nature of the fit. But its recognition is important, for these seizures respond to "Dilantin" and barbiturates, the drugs that control *grand mal*, and the use of diones may well be a waste of time.

The sixth of the minor epilepsies, the psychomotor fits, may be long or quite brief. There is amnesia and loss of contact with environment rather than unconsciousness. There is movement. It may be purposeful but oblivious of surroundings, so that a patient may walk into a wall or undress in public. Or it may be grotesque and bizarre, with grimaces and contortions. Such movement is tonic rather than clonic. These fits are not common in children, but do occur. They may respond to "Dilantin" or barbiturates, but "Phenurone" is said to be the most effective drug.

Finally, there are autonomic fits, sudden exacerbations of some single autonomic function, sudden flushing, abdominal pain, hiccup, gagging, or sudden bursts of rage. The electroencephalogram may be dysrhythmic or normal. The response to drugs is variable, but any anticonvulsant may succeed, and one should be tried after another until the most effective one is found, a principle indeed that applies to any fit that does not respond to the drugs one expects to succeed.

It is plain then that we would be wise to take this vagabond term *"petit mal"* and give it a place of fixed abode. That place I suggest is the true *petit mal* fit with its three cycles per second spike and wave rhythm. Its distinction from myoclonic or akinetic fits is obvious. Its separation from the slower rhythm *petit mal* variant, with so much greater incidence of brain damage, is worthwhile because of the worse prognosis in the latter group. From the point of view of treatment, this spike-wave group can well be kept together, for diones are likely to be the best drugs, and, if they fail, any anticonvulsant can be tried.

However, it is from this very viewpoint of treatment that we should carefully segregate the minor motor and psychomotor fits. In them the hydantoinates and barbiturates are the drugs of choice, and the diones an unpromising last resort.

Reference.

Lennox, W. G., and Davis, J. P. (1950), "Clinical Correlates of the Fast and Slow Spike-Wave Electroencephalogram", *Pediatrics*, 5: 626.

VITAMIN D RESISTANT RICKETS.[1]

By Douglas Galbraith,
Melbourne.

Little did I think when I undertook to give this paper how much I would need to burn the midnight oil; nor how much wear and tear of the cerebral convolutions would be entailed. Frankly, I have found the problems and theories of bone chemistry and physiology in this condition not easy to grasp fully. My hope is that some of my *confrères* will today lend a helping hand, for the subject is certainly a fascinating one.

Causation of Rickets and Osteomalacia.

In its simplest definition rickets and osteomalacia represent a failure to deposit mineral salts in the protein matrix, a failure whose theme song is osteoid tissue and whose causes are various. These include: (*a*) nutritional defects—simple, text-book rickets; (*b*) idiopathic steatorrhœa; (*c*) congenital or acquired idiopathic resistance to vitamin *D*; (*d*) several kinds of kidney disease.

It is not necessary here to discuss the first two group causes. The third, "idiopathic" resistance, is rare. Dr. Mostyn Powell in 1950 described such a condition in several generations of one family. Dr. D. E. Dent, of London, whose work I shall mention in detail later, would perhaps say that this "idiopathic" resistance could have been due to a defect of the renal tubules. In fact he would say that, apart from the rickets occurring from poor vitamin *D* absorption, as in steatorrhœa, all rickets refractory to adequate dosage of vitamin *D* is associated with hitherto unrecognized defects in renal physiology, particularly the tubules.

Dent's views (1952) are convincing and in this paper I propose to follow them somewhat dogmatically.

Rickets due to Kidney Disease.

Dent's classification would be: (*a*) glomerular defect rickets, (*b*) tubular defect rickets.

Glomerular Defect Rickets.

Glomerular defect rickets is classical "renal rickets". The pathological change is mainly in the glomeruli, although the tubules are to a lesser extent affected. Phosphate excretion suffers most, so there is a raised blood phosphate content—in comparison with the lower blood phosphate content of nutritional rickets and of tubular defect rickets.

There is often macroscopic renal pathological change, such as congenital cystic disease or congenital hydronephrosis. Osteoid tissue is scanty because osteoblastic activity is depressed. On the other hand, because of phosphate retention and uræmia, there may be hyperparathyroidism and so bone fibrosis, giving rise to the term renal *osteitis fibrosa cystica*, a term which is misleading and would be better discarded.

Tubular Renal Defect Rickets.

In tubular renal defect rickets the tubules fail to reabsorb phosphate, so there is excessive phosphorus loss, in contradistinction to the phosphorus retention of glomerular rickets. The parathyroids are, of course, unprovoked and so fibrosis of bone does not occur. Osteoid tissue, on the other hand, is freely formed.

According to the particular functions lost by the tubules, various subgroups are distinguished. These are shown in Table I.

It should be noted that in the first four types there is an hereditary tendency, which links them with one another. In distinction, the last two groups are never hereditary, but probably result from acquired infection or kidney toxins. This classification means that Dent rejects the con-

[1] Read at the annual meeting of the Australian Pædiatric Association, Canberra, April 10 to 12, 1953.

TABLE I.

Types of Renal Tubular Defect Associated with Rickets or Osteomalacia.
(After C. E. Dent, 1952.)

Types.	Tubular Resorption Defect.					Tubular Function Defect.		Names Commonly Used.
1	Phosphate.	—	—	—	—	—	—	Resistant rickets, idiopathic osteomalacia. Milkman's syndrome.
2	Phosphate.	Glucose.	—	—	—	—	—	
3	Phosphate.	Glucose.	Amino acids.	—	—	—	—	Fanconi syndrome. Debre - de Toni - Fanconi syndrome. Amindiabetes.
4	Phosphate.	Glucose.	Amino acids.	Water.	Potassium.	Urine acidification.	—	
5	Phosphate.	—	—	—	Potassium.	Urine acidification.	—	Nephro-calcinosis. Butler-Albright syndrome.
6	Phosphate.	—	—	—	Potassium.	Urine acidification.	Ammonia formation.	

ception of vitamin *D* resistance as a cause of rickets or osteomalacia distinct from renal rickets or renal osteomalacia.

It would seem to me that the dividing line between types 5 and 6, on the one hand, and glomerular rickets on the other must sometimes be a fine one; and that as glomerular damage increases and phosphate begins to be retained, the biochemical picture will be almost indistinguishable from that of glomerular rickets. The importance of biochemical control of treatment is obvious.

Treatment of Glomerular Rickets.

Essentially the treatment of glomerular rickets is a question of investigating the pathological condition of the kidneys, particularly for any congenital defect. Since the acidosis is due not only to a shortage of bases, but also to a retention of acid radicles, it is more difficult to correct than is that of tubular rickets.

Administration of vitamin *D* is said to be dangerous, but in the child I shall describe later this has not been so.

Treatment of Tubular Rickets.

It is most important to determine whether or not there is acidosis. In types 1, 2 and 3, in which there is high phosphate clearance without acidosis, the condition will respond to adequate vitamin *D* therapy. Large dosage may be necessary and, as a safeguard, frequent serum calcium investigations must be made.

If acidosis is present it must be corrected by alkali therapy before vitamin *D* therapy will be effective. The leak of bases must be stopped, and until this is done improvement will not occur, no matter how large is the vitamin *D* dosage. If X-ray examination reveals rarefaction a strongly positive calcium balance should be promoted by high calcium intake, citric acid by mouth and extra vitamin *D*. The alkali treatment has to be continued indefinitely. Types 5 and 6 give the most spectacular results with alkali therapy.

Citrate Therapy.—In regard to citrate therapy, Dent thought that its good effect was because it made the gut contents more acid, thus facilitating absorption of calcium. But recently published work by Harold and Helen Harrison, from the Johns Hopkins University School of Medicine (1952), indicates that citric acid without vitamin *D* will cure nutritional rickets and that this effect cannot arise from increased absorption of calcium from the bowel. These authors postulate that the effect of citrate is a local one at the site of the bone lesions, and that it increases the calcifiability of rhachitic bone so that calcium salts are deposited more rapidly than the rate of intestinal absorption of calcium.

A Child with Vitamin D Resistant Rickets.

I want now briefly to tell you of a child who has offered many problems because of her vitamin *D* resistant rickets and who, with alkali therapy, has at last shown bone healing. Even yet there is perhaps a certain doubt as to the exact ætiology.

At the age of eighteen months Sandra, at present aged ten years, appeared to be a normal child. Her early history and family history were quite uneventful.

At the age of three and a half years she began to limp and later was taken to Dr. Charles Richardson, of Ballarat, who diagnosed rickets. Investigation of the blood chemistry at this stage showed a considerably raised phosphate level (9·0 milligrammes *per centum*). Renal investigations gave negative results. In spite of therapy the rickets progressed and became very severe. When she was six and a half years old Dr. Richardson kindly sent her to me and she was admitted to the Children's Hospital Orthopædic Section at Frankston. She was dwarfed and edentulous.

Intensive therapy with vitamin *D* was given, up to 50,000 units of calciferol daily, and for two and a half years there was practically no improvement. About one year ago some slight improvement occurred. Then six months ago alkali treatment was commenced, Shohl's prescription of citric acid and sodium citrate being used. There was great acceleration of healing and today healing at the wrists is fairly complete. For her edentulous condition she was fitted with complete upper and lower dentures and it is interesting that her teeth are now appearing.

Many investigations have been made and we wondered, in particular, about her amino-acid excretion. In this Dr. John Perry has given great help, using the method of paper chromatography. Eventually the amino-acid metabolism was decided to be within normal limits. It is noted, however, by Jonxis, Smith and Huisman (1952) that even in uncomplicated rickets there is often a raised urinary excretion of amino-acids.

The results of the investigations on this patient are as follows.

Renal Tract Investigation.—Pyelography reveals a normal renal tract, and X-ray examination reveals no evidence of calcification.

The urine contains no albumin or sugar; it is microscopically normal. Its specific gravity is low and its reaction alkaline. The total nitrogen content of the urine is normal; its amino-acid nitrogen content is normal. The ratio of amino-acid nitrogen to total nitrogen is normal. Paper chromatography for amino-acids gives normal findings. The urinary calcium content is normal. The urinary phosphate excretion is diminished (0·4 gramme in twenty-four hours, the normal being 1·7 grammes). The urea clearance is 80% of the average.

Investigation of the Blood.—Investigation of the blood yielded the following information: (*a*) The serum calcium content was normal. (*b*) The serum phosphate level was consistently raised (the highest point was 10·0 milligrammes *per centum*). (*c*) The blood urea content was consistently slightly raised (54 to 56 milligrammes *per centum*). (*d*) The serum phosphatase level, originally very high, (126 milligrammes *per centum*), became normal. (*e*) The alkali reserve was consistently below normal (44 volumes *per centum*). (*f*) The serum sodium content was normal. (*g*) The serum potassium level was normal. (*h*) The serum chloride content was normal. (*i*) The blood sedimentation rate was normal.

Summary.—The positive findings are therefore: (*a*) Urinary: (i) Low specific gravity. (ii) Alkaline reaction. (iii) Low phosphate excretion. (iv) Low urea clearance. (*b*) Blood: (i) Raised phosphate content. (ii) Raised phosphatase content. (iii) Raised blood urea content. (iv) Diminished alkali reserve.

Comment.

There is, therefore, acidosis with an alkaline urine, retention of phosphate and impaired renal function.

As I interpret these findings, there must be renal glomerular pathological change and the diagnosis should be renal glomerular rickets. Yet alkali treatment seems undoubtedly to have brought healing—where it should not have done. So it seems likely that, although there is no clear biochemical evidence of this, there is also pathological change in the renal tubules, probably of the Butler-Albright type.

Perhaps part of the mechanism involved is a failure of the proximal tubules to reabsorb bicarbonate from the glomerular filtrate. Latner and Burnard (1950) and Pitts, Ayer and Schiess (1949) have discussed such a mechanism, which could be the one involved in idiopathic renal acidosis of infancy. This could account for the response in this child to alkali therapy.

Bibliography.

DENT, C. E. (1952), "Rickets and Osteomalacia from Renal Tubule Defects", *J. Bone & Joint Surg.*, 34 (B), 266.

HARRISON, H. E. and H. C. (1952), "Further Studies of the Effects of Citrate Feeding on the Calcium, Phosphorus, and Citrate Metabolism of Rachitic Infants", *J. Pediat.*, 41, 756.

JONXIS, J. H. P., SMITH, P. A., and HUISMAN, T. H. J. (1952), "Rickets and Amino-Aciduria", *Lancet*, 2, 1015.

LATNER, A. L., and BURNARD, E. D. (1950), "Idiopathic Hyperchloræmic Renal Acidosis of Infants (Nephrocalcinosis Infantum): Observations on the Site and Nature of the Lesion", *Quart. J. Med.*, 19, 285.

PITTS, R. F., AYER, J. L., and SCHIESS, W. A. (1949), "The Renal Regulation of Acid-Base Balance in Man. III. The Reabsorption and Excretion of Bicarbonate", *J. Clin. Investigation*, 28, 35.

POWELL, M. L. (1950), "Refractory Rickets", M. J. AUSTRALIA, 2, 346.

CONGENITAL OR NEO-NATAL THROMBOCYTOPENIC PURPURA.[1]

By S. E. L. STENING,
Sydney.

PURPURA is a not uncommon symptom in the neo-natal period, being seen, for example, in asphyxia, in hæmorrhagic disease and in hæmolytic disease of the newborn. Purpura in association with thrombocytopenia is much less common.

What is thrombocytopenia? As an arbitrary figure 140,000 platelets per cubic millimetre seems generous as the lower limit of the normal range, but, since Clement Smith gives a range of 140,000 to 290,000 per cubic millimetre, any infant in the first month of life who exhibits purpura or a bleeding state and in whom the thrombocytes are found to number less than 140,000 per cubic millimetre qualifies for the group under discussion.

From a study of available literature, including two critical surveys of the world's literature to 1951, I have been able to find 65 definite examples of congenital thrombocytopenia. I wish, today, to acquaint you with five more examples. (See Tables I and II.)

Classification.

Robson and Walker (1951) have suggested a classification of the types of neo-natal thrombocytopenic purpura. They divide their cases into two groups; the first comprises those infants born of purpuric mothers who suffer from either primary or secondary purpura; and the second, those affected infants born of mothers without purpura or thrombocytopenia.

[1] Read at the annual meeting of the Australian Pædiatric Association, Canberra, April 10 to 12, 1953.

This classification, with the emphasis on the maternal state, seems inadequate, so I should like to present a new classification, which appears to include all cases so far reported. There are three groups.

Group I includes affected infants born of mothers suffering from thrombocytopenia, either essential or symptomatic.

Group II includes affected infants born of normal mothers and without evidence of toxic, poisonous or infective factors in mother or infant.

Group III comprises those infants, born of non-thrombocytopenic mothers, who develop thrombocytopenic purpura in the neo-natal period as a result of some toxic, poisonous or infective factor acquired in their intrauterine or extrauterine existence.

TABLE I.

Observation.	Mother Cases I and II.	Mother Case III.	Mother Case IV.	Mother Case V.
Thrombocytopenia	+	0	0	0
Purpura	+	0	0	0
Splenectomy performed	Yes.	No.	No.	No.
Transfusions ..	Many.	0	0	0
Bone marrow ..	Typical primary thrombocytopenia.	0	0	0
Infection at time of birth of infant ..	0	0	0	0
Other affected children ..	Both.	No.	No.	No.

Group I.

Group I, that of thrombocytopenic infants, born of thrombocytopenic mothers, comprises the only true congenital group, since the symptoms of bleeding in the infant are caused by the passage across the placental barrier of the factor which causes the thrombocytopenic purpura in the mother. The symptoms are self-limited by the eventual expenditure of the factor, though this may take some months, during which time the infant is often dangerously ill or may die.

For example, in Cases I and II the patients were girl infants born at an interval of six years to a mother suffering from primary or essential thrombocytopenia. This mother had had her spleen removed prior to the birth of the elder child. This mother's marrow showed plentiful megakaryocytes but scanty platelet budding. Both infants developed hæmorrhagic symptoms with purpura, rectal bleeding, melæna and hæmaturia in the neo-natal period. Both infants had diminished peripheral thrombocytes, the elder to 11,000 and the younger to 45,000 per cubic millimetre. After treatment mainly by transfusions, both made a recovery and are now normal little girls.

Group II.

Group II comprises affected infants born of normal mothers and without evidence of toxic, poisonous or infective factors in mother or infant. These cases are analogous to the primary thrombocytopenias of later life. Case III is an example of this group.

This male infant was the product of the eleventh pregnancy of a mother with five surviving girls. The mother was free of infection up to the time of appearance of the infant's symptoms. Some days later femoral thrombosis and a possible infection were experienced.

The infant was normal at birth, but purpuric spots were noted between three and four hours after birth. The infant became jaundiced within twenty-four hours, was irritable, had a full fontanelle and crepitations at the lung bases. Blood examination revealed anæmia, no reaction to Coomb's test, 40,000 nucleated red cells per cubic millimetre and about 10,000 thrombocytes per cubic millimetre. He was given an exchange transfusion. Later the thrombocytes disappeared from his circulating blood altogether. Bone marrow examination revealed numerous megakaryocytes with scanty thrombocyte budding. ACTH had a beneficial effect on the hæmorrhagic symptoms, but none on the number of circu-

TABLE II.
Infants.

Observation.	Case I.	Case II.	Case III.	Case IV.	Case V.
Bleeding—					
Skin	+	+	+	+	+
Epistaxis	−	−	−	+	−
Rectal (bright)	+	+	+	+	−
Oral	−	+	+	+	+
Vomitus	−	+	−	−	−
Central nervous system	−	−	+	−	−
Vagina	+	−	−	−	−
Melæna	+	+	+	+	−
Urine	+	+	+	?	?
Conjunctiva	−	−	+	?	+
Infections in infant	0	0	0	0	+
Thrombocytes (lowest number per cubic millimetre).	11,000	45,000	0	Very few or 12,500	110,000
Bleeding time (minutes)	7	17	2	−	−
Coagulation time (minutes)	5½	4	4	−	−
Result of Wassermann test	Negative.	Negative.	Negative.	Negative.	?
Bone marrow	0	0	Primary thrombocytopenia.	0	0
Transfusions	5	3	2 (one exchange)	10	1
Duration of symptoms (days)	93+	30	65	134	8
Result	Normal child.	Normal child.	Slight cerebral damage.	Died.	Normal child.

lating thrombocytes. Recovery occurred after sixty-five days. He appears a normal six-month infant now, but there is the possibility of some cerebral damage.

Case IV was that of an infant born of a mother who suffered from toxæmia of pregnancy and a femoral thrombosis.

He appeared normal for ten days, then there appeared free bleeding from nail scratches on the face. Then bruising of the left eyelid followed by generalized purpura, epistaxis, oral bleeding and melæna. Thrombocytes were absent from the peripheral blood. After a stormy course of 134 days, he died, in spite of ten transfusions. No bone marrow studies were carried out.

Group III.

Group III comprises infants, born of normal mothers, who develop thrombocytopenic purpura in the neo-natal period as a result of some toxic, poisonous or infective factor acquired in their intrauterine or extrauterine existence. These infants correspond to the cases of secondary thrombocytopenia seen throughout all age groups, but are rare in the first month of life. As an example of Group III, I offer Case V.

This infant was the result of a normal third pregnancy. The infant was well for three days, then a red spot was noted on the neck. Soon afterwards, both eyes became swollen and bloodstained discharge appeared in the left eye. A blood count revealed a normal red cell picture, a neutrophile leucocytosis with a shift to the left, and 110,000 thrombocytes per cubic millimetre. This infant recovered in eight days after a single transfusion, *plus* penicillin.

Treatment.

The treatment of these three groups of neo-natal or congenital thrombocytopenia therefore differs. That is why classification is important.

In Group I, in which the infant is affected by the circulating maternal toxin, the treatment is, ideally, replacement transfusion *plus* ACTH or cortisone to control any bleeding tendencies.

Patients in Group II should be treated by supportive transfusions, ACTH or cortisone, and splenectomy if the course is anything but mild.

Those in Group III should be treated by elimination of the toxic, infective or poisonous cause, *plus* supportive transfusions.

In conclusion, may I stress the fact that, though the number of cases of neo-natal thrombocytopenia in the literature is small, these cases are not uncommon.

Reference.

ROBSON, H. N., and WALKER, C. H. M. (1951), "Congenital and Neonatal Thrombocytopenic Purpura", *Arch. Dis. Childhood*, 26, 175.

Addendum.

Since this paper was prepared, I have seen yet another case, which must go into Group III. It was a severe case of *icterus gravis neonatorum* treated by replacement transfusion. The infant had petechiæ on the face within a few hours of birth. Later there was rupture of the bowel wall at the site of petechial hæmorrhages, with ileus and peritonitis. This infant had two counts of 30,000 and 20,000 thrombocytes per cubic millimetre before her death.

RECENT TRENDS IN THE DIAGNOSIS AND TREATMENT OF CŒLIAC DISEASE.[1]

By CHARLOTTE M. ANDERSON,
Melbourne.

MODERN interest in cœliac disease began with Samuel Gee's description in 1888, and the nature and treatment of the condition have been the source of much discussion ever since. That there is a defect in the intestinal absorption mechanism has been largely accepted, but the nature of this was unknown, and treatment was directed towards dietary modification and general supportive measures to combat the specific nutritional deficiencies appearing during the course of the disease.

Until recently dietary treatment was of two main types —the diet with a low fat content recommended by Sir Leonard Parsons and others, and the complex carbohydrate and cereal-free diets of many American workers, notably Howland, Haas, Andersen, May and Lowe. These diets were also high in protein content, and some workers, especially Haas, recommended bananas. In 1948 this régime was put forward as the "starch-free" diet by Sheldon, of Great Ormond Street. This latter type of régime was moderately successful although difficult to attain, and the reason for its success will be appreciated later.

Working quite independently in Holland during the 1940's, Dicke observed that cœliac children under his care could tolerate all food with the exception of wheat and rye flour. Together with Weijer's and Van de Kamer, he was able to show by very comprehensive and careful clinical and fat balance studies that clinical improvement and a decrease of fæcal fat occurred when wheat and rye flour were excluded from the diet, and that the reintroduction of wheat flour led to deterioration.

During the past two years I have been associated in Birmingham, England, with a team of workers led by

[1] Read at the annual meeting of the Australian Pædiatric Association, Canberra, April 10 to 12, 1953.

Professor A. C. Frazer, who for some years has been studying the mechanism of normal and defective fat absorption. With the cooperation of Professor Smellie, of the Department of Child Health, we were able to include in this study a group of children suffering from cœliac disease.

The object of our work has been twofold—firstly to study the gastro-intestinal changes in a group of cases of cœliac disease using methods by which Frazer and colleagues had demonstrated the absorption defect in adult idiopathic steatorrhœa, and secondly to study these changes under different dietary régimes in order to confirm and possibly to extend the work of Dicke.

The cases were diagnosed according to the classical clinical features of the disease—failure to thrive of insidious onset in late infancy and early childhood, anorexia, temperamental difficulties, failure to gain or loss in weight, this going on to wasting, large pale offensive stools, protuberant abdomen and usually hypochromic anæmia.

The diagnosis was confirmed by the application of the following tests.

1. Fat absorption by fat balance. Fæcal fat was estimated daily by a wet extraction method (Anderson, Frazer *et alii*, 1952), the biochemical technique of which is simple and rapid. With the child on a known adequate intake of fat, 40 to 46 grammes daily depending on age, over 90% of the fat should be absorbed. In a case of cœliac disease 60% to 85% is the usual figure.

2. Oral glucose tolerance curve. This was used as a measure of the absorption of glucose and in cœliac disease the curve is flattened (less than 40 milligrammes *per centum* rise) to a greater or lesser degree depending on the severity of the condition at the time.

3. Chylomicrograph used as a measure of the post-absorptive lipæmia. Fat particles in serum were counted, dark ground microscopy being used after a fatty meal and a curve being constructed. The lipæmia is delayed and depressed.

4. Duodenal intubation. The enzymes, amylase, lipase and trypsin were present in normal amounts in this series.

5. Barium X-ray studies of small bowel with two types of barium (Anderson, Astley *et alii*, 1952). (*a*) Simple barium and water gave a "clumped" pattern (also obtained in normal children). (*b*) "Non-flocculating" or "suspended" barium showed the small bowel loops to be dilated and probably atonic, with a barred appearance due to the parallel transverse mucosal folds (*valvulæ conniventes*).

The changes observed in these tests are identical with those found in adult idiopathic steatorrhœa and indicate that there is an absorption defect in cœliac disease, certainly affecting fat and carbohydrate, and that the small bowel is dilated and probably atonic.

The effect of the removal and reintroduction of wheat flour into the diet of a group of patients was studied with these methods, in conjunction with change in clinical condition and weight.

A typical case was that of a girl, aged three years, who had been ill for nine months with diarrhœa, intermittent vomiting and progressive emaciation. She weighed sixteen pounds and was unable to stand. She was placed on a diet free of wheat or rye flour, but was allowed to eat anything else she desired. In two weeks her appetite and temperament had improved considerably and in three to four weeks stools were becoming formed. In ten weeks there was a ten pound gain in weight. At the end of this time the daily fæcal fat content, the chylomicrograph reading, the oral glucose tolerance curve and the X-ray appearance of the small bowel had returned to normal.

After the patient had been on the wheat-free diet for three months wheat flour was introduced into the diet. The result was followed by means of a continuous fat balance record. On a 40 gramme daily fat intake the output was less than four grammes daily. After the inclusion of wheat flour, clinical relapse commenced one week later, firstly with a decrease in appetite and increase in irritability, and then with recurrence of diarrhœa and increase in fat content of the stools up to 10 to 15 grammes daily.

After forty days, wheat flour was replaced by wheat starch. All cereals are made up of starch and a protein component, which in the case of wheat and rye is known as gluten. In this case the starch portion was used and gradual improvement in fat absorption occurred, again associated with clinical improvement. This was maintained until wheat flour was again introduced, this time non-agenized flour, when there was a deterioration of similar nature.

In all, fifteen cases were studied in this way, and in each case after six weeks on a diet containing no wheat flour the stools had become normal in character and fat content, concomitant with clinical improvement which tended to take place more rapidly than the recovery to normal of the stools.

As these findings together with those of Dicke's had shown that the starch portion of wheat flour could be well tolerated, it seems as though the factor causing deterioration was confined to the gluten fraction. This was confirmed in a group of cases by similar continuous fat balance studies. After improvement had taken place on a diet containing no wheat flour, and the fat absorption was normal, gluten was fed to the children, and the same clinical and biochemical deterioration was observed.

In all cases studied, when fat absorption became normal there was also a return to normal in the chylomicrograph reading, in the glucose tolerance test curve and lastly in the X-ray picture of the small bowel, with similar deterioration in the results of these tests after the feeding of gluten.

The precise way in which gluten brings about an absorption defect in this group of children and not in normal children is as yet unknown. However, the contribution of the Dutch workers has permitted a very much simplified and more effective way of managing this troublesome condition to be worked out.

The child may eat a normal diet, provided all foods made with wheat or rye flour are excluded. Attached is a recommended dietary régime which could be given to the parents. (See Addendum.) It can be seen that there is a wide range of choice of foods.

In our experience reduction in fat intake is unnecessary, except rarely in the first few weeks of treatment in more severe cases. Adequate calorie intake is important as well as a balanced diet.

All patients have remained well on the diet, and only infrequent visits to hospital for check-up are necessary. However, until we know the way in which gluten is acting, it is recommended that the dietary restriction be maintained throughout the growing period of the child, as there is already sufficient evidence from follow-up experiments to conclude that cœliac children do not reach normal adult development if untreated, and some at least do become adult sufferers from idiopathic steatorrhœa.

References.[1]

ANDERSON, C. M., FRAZER, A. C., FRENCH, J. M., GERRARD, J. W., SAMMONS, H. C., and SMELLIE, J. M. (1952), "Cœliac Disease: Gastro-intestinal Studies and the Effect of Dietary Wheat Flour", *Lancet*, 1: 836.

ANDERSON, C. M., ASTLEY, R., FRENCH, J. M., GERRARD, J. W. (1952), "The Small Intestine Pattern in Cœliac Disease", *Brit. J. Radiol.*, 25: 526.

Addendum.

Wheat and Rye Gluten-free Diet for Cœliac Disease.

The child may eat a normal diet with the exception of any food made with wheat or rye flour. These flours contain a substance—gluten—which is harmful to the child. When wheat flour is specially treated to remove gluten, the starch portion is left behind and can be used in cooking. This "special wheat starch" can be obtained from most starch and cornflour manufacturers. (Inquiries should be made from the main firms in each State.) Special starch, cornflour, or soya flour, or a mixture of these can be used to make special biscuits, cakes, pastries and tea loaf. Cornflour is used to thicken gravies, stews, custards and sauces.

For breakfast cereals, "Cornflakes", "Rice Bubbles" or oatmeal porridge are satisfactory.

Foods allowed are: all meats and fish, whole milk, fruits, vegetables, potatoes, rice and oats, butter and cheese, other fats, eggs and sugars.

[1] Details of methods, graphs and tables are set out in the two papers mentioned.

Foods to be avoided are: bread (any commercial brand), biscuits, pastries, cake; breakfast cereals such as "Weeties", puffed wheat; macaroni, vermicelli and spaghetti; "Ry-vita" and rye bread; sausages (bread-containing); tinned meats and soups containing wheat flour; "Farex", "Benger's Food", custard powders and "Ovaltine".

Example of a wheat and rye gluten-free diet:

Breakfast.—"Cornflakes", "Rice Bubbles" or oatmeal porridge; sugar and whole milk; boiled or poached egg, bacon and tomato; wheat starch, cornflour or soya flour biscuits or loaf; butter; "Marmite" or honey.

Mid-morning.—Fruit, orange juice or milk.

Luncheon.—Salad, cheese, tomato or egg; special biscuits or loaf; butter; milk; cornflour custard and stewed fruit or raw fruit.

Mid-afternoon.—Cadbury's cocoa or "Bournvita"; milk; special cake or biscuit.

Dinner.—Average portion of meat, chicken or fish; gravy thickened with cornflour; potato and vegetables; dessert such as rice or sago custard, cornflour blanc mange, jelly, Spanish cream, baked or boiled milk and egg custard; stewed fruit.

Recipes—Gluten Free.

Tea Loaf.—1 cup cornflour or wheat starch, 1 tablespoon butter, 1 egg, 1 teaspoon baking powder, essence of lemon, ½ cup sugar, ½ cup milk.

Method. Rub butter into flour, add sugar and then make into a dough with egg and milk. Cook half to three-quarters of an hour in a moderate oven.

Biscuits.—3 ounces cornflour or wheat starch, 3 ounces soya bean flour, 4 ounces sugar, 1 egg, 3 ounces margarine, vanilla or orange rind.

Method. Cream the margarine and sugar, add the egg and beat again. Sift cornflour and soya bean flour and add. Lastly, add a few drops of vanilla or orange flavouring. knead well, roll out to quarter of an inch thickness, cut into shapes and bake in moderate oven for ten to fifteen minutes.

Special Wheat Starch Bread Loaf.—Four ounces wheat starch, pinch of salt, half an ounce of sugar, half an ounce of butter or margarine, one and a half ounces of soya flour, one teaspoon malt extract, one level teaspoon cream of tartar, half a level teaspoon bicarbonate of soda, one egg, two tablespoons milk.

Method: Sift starch, salt, soya flour, cream of tartar and soda. Rub in fat, add sugar and mix into a moist dough with beaten egg and milk and malt extract. Place in a greased nut loaf tin or small cake tin and bake in a moderately hot oven thirty-five to forty-five minutes. This recipe has been found to be much easier for mothers to make and more palatable for the child.

URINARY EXCRETION OF MERCURY IN PINK DISEASE: PRELIMINARY REPORT.[1]

By F. W. CLEMENTS,
Sydney.

PREAMBLE.

WITHIN recent years a number of writers have suggested that mercury may be an ætiological factor in pink disease. Overseas investigations have been inconclusive, prompting W. S. Craig to suggest that the significance of mercury as an ætiological factor must be looked upon as *sub judice*.

The preliminary reports cover the results of the analyses of the urine from 180 infants for mercury content. The subjects came from two sources. (*a*) Apparently healthy, normal infants attending baby health centres. (These specimens of urine were obtained through the courtesy of Dr. Grace Cuthbert-Browne.) (*b*) Patients in the Royal Alexandra Hospital for Children. (These specimens of urine were obtained through the courtesy of the Medical Superintendent and the honorary medical staff.) The analyses were made by Frank Barrett, M.Sc., of the staff of the School of Public Health and Tropical Medicine, Sydney. The method used was a modification of that developed by Lang and Nelson, which, in Barrett's hands, gives approximately 100% reproducibility for low concentrations and about ±5% variation for high concentrations.

[1] Read at the annual meeting of the Australian Pædiatric Association, Canberra, April 10 to 12, 1953.

INFANTS ATTENDING BABY HEALTH CENTRES.

The urine from 137 infants attending baby health centres in Sydney suburbs has been examined. When the samples were collected information was also obtained about the consumption of teething powders including the brands, number of powders given over a period of time and the date on which the last powder was given. We also asked for information about the use of ointments, prescriptions were asked for, and the general health of the infant was investigated.

TABLE I.

Frequency Distribution of Samples of Urine from Infants Attending Baby Health Centres.

Concentration of Mercury.	Number of Infants.
Less than 2 microgrammes per 100 millilitres	119
2 to 4 microgrammes per 100 millilitres	9
5 to 9 microgrammes per 100 millilitres	2
10 to 19 microgrammes per 100 millilitres	2
20 to 29 microgrammes per 100 millilitres	3
38 microgrammes per 100 millilitres	1
240 microgrammes per 100 millilitres	1

Mercury Concentration in Urine from 137 Infants.

Table I gives the frequency distribution of various concentrations of mercury in the urine from 137 patients.

The health of all infants who had excreted mercury in the urine was checked at intervals of one to two months after the date on which the sample was collected. So far all have remained healthy.

Relationship of Urinary Excretion of Mercury to Ingestion of Teething Powders.

There are four common brands of teething powders on the market in Sydney, but in addition a number of pharmacists dispense their own products. Two of the common brands contain mercury, one 0·3 grain of calomel, and the other 0·7 grain, per powder. The other two brands are apparently 100% lactose. Some of the powders dispensed by pharmacists contain mercury, others do not.

The History of the Administration of Teething Powders to Children Not Excreting Mercury in the Urine.

The urine from 119 infants did not contain mercury or contained 1·0 microgramme per 100 millilitres. Table II shows the relevant facts about the consumption of teething powders as told by the mothers.

The frequency of administration varied widely, as Table III shows.

The frequency of administration of teething powders containing mercury was as follows:

Child 1. Two only about one month before.
Child 2. Two only about three weeks before.
Child 3. One about six weeks before.
Child 4. One about one month before.
Child 5. One about two weeks before.
Child 6. One about two weeks before.

It will be seen that the children who had been given mercury-containing teething powders had taken them at least two weeks prior to the collection of the specimen and thus had had an opportunity to excrete the mercury before the date of the test.

History of Administration of Teething Powders to Children Excreting Mercury in Urine.

The mothers of 17 of the 18 children who excreted mercury admitted to giving the child a mercury-containing teething powder up to within a few days before the collection of the urine. For most children the dose was small, being of the order of one powder occasionally, but for the children who excreted 38 microgrammes per 100 millilitres and 240 microgrammes per 100 millilitres the dosage was relatively large, being three packets of powders weekly for the latter. In general, the amount excreted in the urine bore a close relationship to the amount said to have been given to the child.

Table II.

Consumption of Teething Powders by Infants Attending Baby Health Centres.

Group.	Number of Infants.
No teething powders	82
Teething powders not containing mercury	31
Teething powders with mercury ..	6
Total	119

No child in this series had been treated with a mercury-containing ointment.

Patients in the Royal Alexandra Hospital for Children.

Specimens of urine were collected from three types of patients at the Royal Alexandra Hospital for Children.

Table III.

Frequency of Administration of Teething Powders not Containing Mercury.

Administration.	Number of Infants.
Three times a day	1
Twice daily	3
Once a day	8
When needed	2
Occasionally	12
Rarely	5
Total	31

1. Controls. Patients suffering from a variety of diseases other than pink disease or nephritis, of whom there were 21.

2. Patients suffering from nephritis or nephrosis, of whom there were 11.

3. Patients admitted to hospital with the diagnosis or provisional diagnosis of pink disease, of whom there were 10.

As with the infants attending the baby health centres, an attempt was made to collect information about the intake of mercury or the use of mercury ointments.

Results.

Controls.

Of the 21 controls, 12 excreted less than two microgrammes per 100 millilitres of urine; one excreted two to four microgrammes per 100 millilitres of urine; five excreted from five to nine microgrammes per 100 millilitres of urine; one excreted from 10 to 19 microgrammes per 100 millilitres of urine; one excreted 28 microgrammes per 100 millilitres of urine; and one excreted 34 microgrammes.

Nephritis, Nephrosis.

Six patients with nephritis or nephrosis excreted less than two microgrammes of mercury per 100 millilitres of urine.

Five other patients had the following records.

Patient 1, excreting 3 microgrammes, had been given teething powders.

Patient 2, excreting 15 microgrammes, had been given teething powders.

Patient 3 gave three readings at intervals, 50, 10 and 2 microgrammes. Teething powders, many of which contained mercury, had been given for nine months.

Patient 4, excreting 11 microgrammes, had been given an injection of an organic mercurial preparation.

Patient 5 gave three readings, 5, 11 and 45 microgrammes, at intervals following the injection of an organic mercury preparation.

Pink Disease.

Ten children had been admitted to hospital with the provisional diagnosis of pink disease. The history of each patient was carefully studied and discussed with the honorary medical officer without either his or my knowledge of the results of the urinary analysis. We found that the patients could be placed in one of three categories —"pink disease", "probably pink disease" and "not pink disease".

Pink Disease.—These children had most if not all the clinical features detailed by Southby in his recent article. There were five patients in this category and the relevant facts about each are set out below.

1. A female patient, aged fourteen months, had been given teething powders for nine months, mostly mercury-containing, 24 in the two months preceding the first estimation. The mercury in the urine on September 30, 1952, was 48 microgrammes per 100 millilitres of urine; on October 30, 1952, the amount was 12 microgrammes.

2. A male child, aged nine months, was given teething powders containing mercury once a week as a laxative from the age of four months; the dosage was increased to one a day during teething; the most recent increase had been for two weeks which ended two weeks prior to the first estimation. The mercury in the urine on February 11, 1953, was 49 microgrammes per 100 millilitres, and on February 28, 1953, the amount was 38 microgrammes per 100 millilitres.

3. A male child, aged nine months, had been given mercury-containing teething powders regularly (about twice weekly) in the three months preceding the first estimation. The amounts of mercury in the urine were as follows: on November 7, 1952, 49 microgrammes per 100 millilitres; on November 19, 1952, 32; on November 26, 1952, 36; and on November 27, 1952, 14 microgrammes. This infant was given BAL following the estimation of November 19, 1952.

4. A male child, aged two years, had been taking mercury-containing teething powders regularly (about one weekly) for about a year. His urine on November 26, 1952, contained 21 microgrammes of mercury per 100 millilitres; on November 11, 1952, it contained 10 microgrammes, and on February 25, 1953, nil.

5. A male child, aged fourteen months, had been given mercury-containing teething powders for two months, the last fourteen days before estimation. The mercury in the urine was 11 microgrammes per 100 millilitres.

Probably Pink Disease.—Two patients were thought to be recovering from pink disease.

1. A male child, aged twenty-four months, had been given 48 teething powders containing mercury since he was nine months of age. The mercury in his urine was three microgrammes per 100 millilitres.

2. A female child, aged nineteen months, had been given mercury-containing teething powders irregularly. The urine contained six microgrammes of mercury per 100 millilitres.

Not Pink Disease.—In three cases the history, clinical features and results of laboratory tests denied pink disease. One patient thought to be suffering from urticaria had no mercury in the urine. In one case marked "for investigation" the urine contained 1·4 microgrammes of mercury per 100 millilitres. In one case of upper respiratory tract infection the amount of mercury was four microgrammes per 100 millilitres.

DISCUSSION.

These investigations, to date, have revealed nothing new —they have certainly not proven anything. The main features are:

1. All patients suffering from pink disease were excreting relatively large amounts of mercury in the urine.

2. Several apparently healthy infants excreted comparable amounts of mercury, one infant a much greater amount.

3. The patients with pink disease had been given mercury-containing teething powders for relatively long periods prior to admission to hospital. In contrast, although one apparently normal infant had been given a large dose of mercury, the administration had been for a short period just prior to the examination of the urine. This is an aspect of the problem that we intend to follow up in the future.

As a side issue it is important to realize that at least one third of infants and children in this study had been given teething powders. Some were given one brand only; others a variety. A check of the number of times each brand was mentioned in the histories showed that teething powders without mercury were mentioned 35 times and those with mercury on 22 occasions.

This ratio roughly follows the distribution on the market, so it seems almost chance or perhaps the effects of advertising that determines which teething powders a child is given.

POLIOMYELITIS VIRUS IN TISSUE CULTURE.[1]

By J. R. THAYER,
Commonwealth Serum Laboratories, Melbourne.

THE discovery by Enders and his co-workers in 1949 that poliomyelitis virus would grow in flask or test tube cultures of human non-nervous tissue, followed by the observation of the degeneration of tissue cells that usually paralleled the growth of the poliomyelitis virus, and then the work of Syverton *et alii* in 1951 demonstrating the suitability of monkey testicular tissue for such technique, greatly expanded the areas of research in poliomyelitis. Until this time the use of whole living monkeys, expensive animals to purchase and maintain, was of prime importance in poliomyelitis research, although much valuable work had been carried out with the mouse-adapted Lansing strain.

Tissue culture methods of handling poliomyelitis virus were soon adopted in a number of American laboratories, and work, initiated by Dr. P. L. Bazeley with the concurrence of Dr. F. G. Morgan, was commenced at the Commonwealth Serum Laboratories in Melbourne. Early workers made use of biochemical changes, particularly that of pH, as an index of poliomyelitis virus growth, such results being confirmed by monkey or mouse passage. However, such methods left much to be desired and the specific degenerative changes which were noted gave the required impetus to the project.

Briefly, the principle involved is as follows. In actively growing tissue culture new fibroblastic outgrowth occurs. The addition of living poliomyelitis virus to the tissue culture causes degenerative changes in the fibroblasts which can be observed under the microscope. Finally, these degenerative changes may be prevented by the addition of specific serum.

The tissue used must be human or monkey tissue. At first embryonic tissue was considered essential, but many adult tissues, such as skin or testes, will suffice, although not all tissues will give a satisfactory outgrowth or support poliomyelitis virus growth. The tissue outgrowths obtained under test tube conditions are predominantly or entirely fibroblastic. Monkey testicular tissue is fairly readily obtainable, gives satisfactory consistent fibroblastic outgrowth which supports the growth of poliomyelitis virus with characteristic cytopathogenic effect and is used as a routine.

The monkey testicular tissue is handled on the day of collection. We have had little success to date with methods of preserving it. It is cut by scalpel into pieces about 1·0 millimetre square which are then fastened upon the inside of a suitable container (we use 20 millilitre penicillin bottles) by means of a plasma clot and the nutrient fluid is pipetted in. The medium is a balanced salt solution containing horse serum, chick embryo extract and the antibiotics streptomycin and penicillin.

After incubation of the tissue at 36° C. for five or six days in stationary bottles, we have not found it necessary to use roller tube cultures; there is usually a satisfactory outgrowth of fibroblasts. All bottles are checked microscopically by the use of a plate microscope of 32 magnifications for such outgrowth before being used further. We are now ready to inoculate the tissue with poliomyelitis virus or with specimens suspected to contain virus. The culture medium is removed, the virus inoculum pipetted into the bottle and this is followed about ten minutes later by fresh culture medium.

The bottles are reincubated, still stationary, and are examined, unstained, four to eight days later with a low power of the microscope for the specific cytopathological change induced by the poliomyelitis virus. The earliest degenerative changes occur at the periphery of the fibroblastic outgrowth, where we find patches of cells containing coarse cytoplasmic granules. These areas spread and coalesce, giving diffuse granularity. Varying degrees of cytolysis follow, with loss of the highly refractile appearance of the nucleus and diminution in the size of the fibroblastic outgrowth.

The general principle, therefore, is that tissues are grown for five or six days and then inoculated, and the presence or absence of poliomyelitis virus is shown by the presence or absence of specific cytopathogenic effect four to eight days later. This principle may be used in various ways:

1. To isolate virus from fæces, spinal cord *et cetera*. From fæces we make a 20% suspension in sterile distilled water, centrifuge out the debris at high speed, add high concentrations of streptomycin (5000 microgrammes per millilitre) and penicillin (1000 units per millilitre) and use this fæcal extract to inoculate the tissues.

2. To type poliomyelitis viruses. When cytopathogenic effect is observed we take the tissue culture supernatant containing virus and test for neutralization of cytopathogenic effect by means of antisera specific to the various poliomyelitis virus types—Type 1 (Brunhilde), Type 2 (Lansing) and Type 3 (Leon).

3. To titrate poliomyelitis antibodies in sera. Here we use standard strengths of poliomyelitis type strains and determine the highest serum dilution which will neutralize the cytopathogenic effect of each virus. In the light of recent work on the production of serum antibodies to poliomyelitis, the application of this technique to routine serum diagnosis of poliomyelitis has to be revised.

4. To grow poliomyelitis virus in bulk in tissue culture and to produce a vaccine which may be used prophylactically. Active work on this project is being carried out by Dr. P. L. Bazeley, now in America, and prospects of producing a useful and safe vaccine are considered good. At first it appeared that an attenuated live vaccine was possible, because the animal virulence of poliomyelitis strains diminishes as they are passaged in tissue culture, but use of a formalin inactivated virus vaccine now appears more probable, particularly since the development of non-protein media for the growth of tissue cultures.

The tissue culture unit at the Commonwealth Serum Laboratories has been used as an experimental project to investigate and make trial of various techniques and no attempt has been made to use it for purposes of routine

[1] Read at the annual meeting of the Australian Pædiatric Association, Canberra, April 10 to 12, 1953.

diagnosis. It is of interest, however, that during 1951 in Melbourne no Type 2 (Lansing) strains were detected. Of 34 fæcal specimens from cases of poliomyelitis in Fairfield, 15 specimens yielded Type 1 (Brunhilde), two specimens Type 3 (Leon) and one specimen both Type 1 and Type 3.

Since late 1952 until the present time we have been unable to find a single Type 1 strain, but Type 2 strains and a few Type 3 strains have been isolated from metropolitan and country areas in Victoria and from Hobart. It is of interest to note that the isolation rate of Type 2 and Type 3 virus strains in the current epidemic has fallen far short of the approximate 50% isolation rate in the previous Type 1 epidemic. One of the biggest problems in tissue culture yet to be solved is the fact that some poliomyelitis strains, especially Type 2 and Type 3 strains, may grow in tissue culture, but not produce visible evidence of their growth—that is, some strains are non-cytopathogenic. Experimental work with mice is now in progress and may yield more evidence regarding the true incidence of Type 2 strains in the material we are handling at the moment.

In concluding, I wish to thank Dr. F. G. Morgan, of the Commonwealth Serum Laboratories, for making possible the presentation of this paper. Although it is necessarily a brief outline, much detailed procedure having been omitted, I believe that in tissue culture technique we possess a tool which, while still in its early experimental stage, has in the near future bright hopes of yielding answers in the diagnostic field. We also have the far more important prospect of providing a worthwhile inactivated poliomyelitis vaccine.

COXSACKIE VIRUS INFECTIONS IN AUSTRALIA, WITH SPECIAL REFERENCE TO THEIR EPIDEMIOLOGY.[1]

By N. F. STANLEY,
Sydney.

IT is proposed to describe briefly the nature of the Coxsackie viruses that have so far been studied, the human diseases with which they are associated, and epidemiological data obtained in Sydney and the Western Pacific area.

The Coxsackie viruses may be divided into two groups, A and B, depending to a large extent on the lesions produced as a result of their infecting suckling mice, the nature of the syndrome of infection, and the incubation period. In Sydney, evidence has been obtained for the existence of a third group (Stanley *et alii*, 1953), which is similar in some respects to the group B viruses. The group A viruses produce mainly destruction of the skeletal muscle in infant mice, whereas the group B viruses produce lesions in the central nervous system and focal myositis is difficult to demonstrate. All the Coxsackie viruses are highly resistant to germicides, such as lysol, alcohol, phenol, ether and all the antibiotics. They may be killed quickly only with formalin or by heating. (See Table I.)

Human Disease.

Group A Viruses.

The first observation of any importance on the natural occurrence of Coxsackie viruses came from Maryland, United States of America (Huebner *et alii*, 1952), and ultimately led to the association of group A infections with the condition of "herpangina", which is a summer illness of children consisting of fever and sore throat with vesicular and ulcerated lesions on the anterior tonsillar pillars or soft palate. Group A viruses of different antigenic types were isolated from 36% of patients with herpangina, whereas the virus could be isolated from only 1% of a control group, comprising 992 persons.

[1] Read at the annual meeting of the Australian Pædiatric Association, Canberra, April 10 to 12, 1953.

Group B Viruses.

Evidence is accumulating for the association of group B Coxsackie infections with Bornholm disease. This association has been demonstrated in the United States of America by Dalldorf, Lazarus *et alii*, in England by Findley *et alii*, and Brown *et alii* (1952), in Switzerland by Thelin and Wirth (1951), and in New South Wales and Tasmania by Stanley and Dorman (1953). More recently still, a group B virus was isolated from a muscle biopsy specimen during the acute phase (Lepine *et alii*, 1952). To my knowledge this has not yet been confirmed.

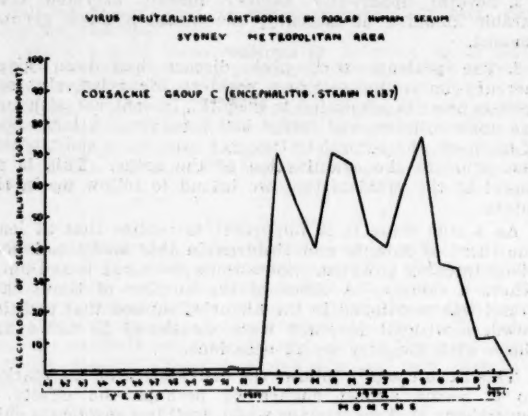


FIGURE I.

Group C Viruses.

In January, 1952, there was an explosive outbreak of mild encephalitis in the Sydney metropolitan area, and there were no fatal cases. We subsequently showed that this outbreak was associated with a group of Coxsackie viruses, all antigenically homogeneous and differing in some respects from any Coxsackie virus yet described, although it is related to group B, type 3. A temporary designation of group C was given to these infective agents. In a communication I have just received from Dr. Dalldorf, Director of the New York State Department of Health, he

TABLE I.

A Comparison of the Properties of Coxsackie Viruses Groups A and B with the Newly Described Group C.

Observation.	Coxsackie Group.		
	A.	B.	C.
Myositis	+	+	−
Encephalitis	−	+	+
Incubation period (days)	2–3	3–8	5–17
Serological type	Numerous.	Few.	One.
Symptoms of infection in suckling mice.	Paralysis, spasticity *et cetera*. (Die quickly.)	Tremors, emaciation, wrist drop. (Die slowly.)	Tremors, emaciation, wrist drop. (Die slowly.)
Association with disease in man.	Herpangina, ? poliomyelitis.	Bornholm disease.	Encephalitis.
Resistance to germicides	Markedly resistant to lysol, phenol, alcohol, ether, *et cetera*.	Resistant to lysol, phenol, alcohol, ether *et cetera*.	Sensitive to lysol, phenol, 70% alcohol.
Age susceptibility of mice	Up to 5 days.	Up to 5 days.	Up to 1 day.

confirms our views as to this being a distinct nosological entity, and he is at the moment carrying out further tests with the virus strains which we sent him. The virus was isolated from the fæces of acutely ill patients, and neutralizing antibodies were demonstrated in their sera to high titre. By indirect epidemiological means we estimated that at least 100,000 individuals were infected with the virus and developed protective antibodies over a period of a few weeks.

Poliomyelitis.

Much more evidence is needed in the study of any association that may exist between Coxsackie infections and poliomyelitis. Coxsackie viruses have been isolated

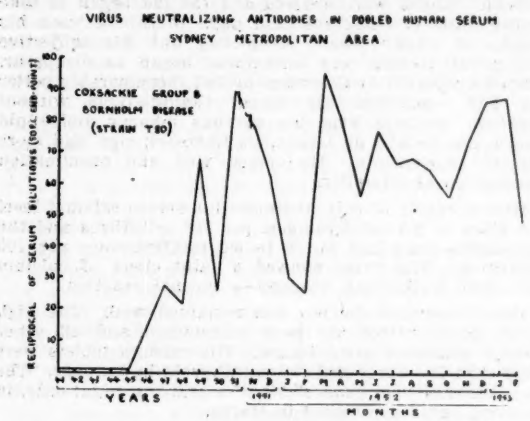


FIGURE II.

from the fæces and throat washings of patients with paralytic and non-paralytic poliomyelitis either alone or together with a poliomyelitis virus. The variation in the findings is considerable. The Easton epidemic of poliomyelitis (described by the Yale School) is of particular interest because of the high ratio of paralytic to non-

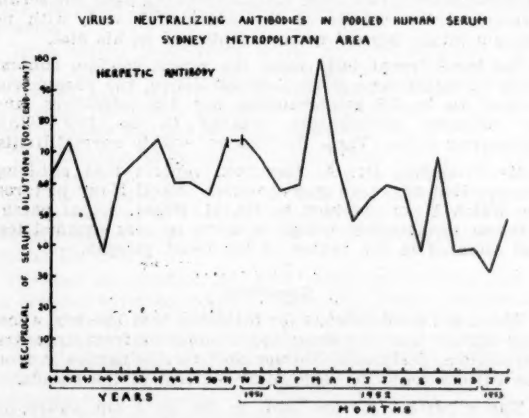


FIGURE III.

paralytic cases, the high proportion of fatal cases and the isolation of both poliomyelitis and group A Coxsackie viruses from more than half of the patients selected. Similar findings have not been forthcoming with group B viruses. I have not the time to discuss the considerable amount of work we have done in attempting to demonstrate interference between poliomyelitis viruses and group B Coxsackie viruses.

Epidemiology.

Because Coxsackie viruses are relatively resistant viruses and because of the widespread distribution, their recovery from raw sewage is relatively easy. We have recovered many strains in this way, and they may give some idea of the predominant infective agents that exist each month. Although this may be true, we have obtained information of greater value by studying the antibody response to infection both of individuals and of pooled human serum. I have said very little about the estimation of Coxsackie antibody, but we have been using a very sensitive test in the suckling-mouse neutralization test. Neutralizing or protecting antibodies in pooled human serum collected in the Sydney metropolitan area were shown in graphs (See Figures I, II and III.) This method also shows that group A and group B strains are widely distributed throughout the Western Pacific area from Tasmania to Korea, and from Western Australia to Fiji.

References.

BEEMAN, E. A., COLE, R. M., and HEUBNER, R. J. (1952), "Studies in Man of Neutralizing Antibodies against Group A Coxsackie (Herpangina) Viruses. Prevalence of Antibodies in a Community in 1949 and 1950", *Am. J. Hyg.*, 56: 215.

BEEMAN, E. A., and HEUBNER, R. J. (1952), "Evaluation of Serological Methods for Demonstrating Antibody Responses to Group A Coxsackie (Herpangina) Viruses", *J. Immunol.*, 68: 663.

BROWN, C. M., LIDDLE, D. C., and TOBIN, J. O'H. (1952), "Bornholm Disease and Coxsackie Viruses", *Lancet*, 1: 445.

FINDLAY, G. M., and HOWARD, E. M. (1950), "Coxsackie Viruses and Bornholm Disease", *Brit. M. J.*, 1: 1233.

LAZARUS, A. S., JOHNSTON, E. A., and GALBRAITH, J. E. (1952), "An Outbreak of Epidemic Pleurodynia, with Special Reference to the Laboratory Diagnosis of Coxsackie Virus Infections", *Am. J. Pub. Health*, 42: 20.

LEPINE, P., DESSE, G., and SAUTTER, V. (1952), "Histological Examination and Isolation of Coxsackie Virus from Muscle Biopsy Material of Cases of Epidemic Myalgia (Bornholm Disease)", *Bull. Acad. Nat. Méd.*, 136: 66.

MELNICK, J. L., KAPLAN, A. S., ZABIN, E., CONTRERAS, G., and LARKUM, N. W. (1951), "An Epidemic of Paralytic Poliomyelitis Characterized by Dual Infections with Poliomyelitis and Coxsackie Viruses", *J. Exper. Med.*, 94: 471.

STANLEY, N. F., HAYES, L., and DORMAN, D. C. (1951), "Suckling Mouse Viruses in New South Wales: Their Relationship to the Coxsackie Group of Viruses", *Australian J. Exp. Biol. & M. Sc.*, 29: 367.

STANLEY, N. F., and DORMAN, D. C. (1953), "Group A Coxsackie Viruses Isolated from Cases of Poliomyelitis", *Australian J. Exper. Biol. & M. Sc.*, 31: 9.

STANLEY, N. F., DORMAN, D. C., and PONSFORD, J. (1953), "A Hitherto Undescribed Group of Coxsackie Viruses Associated with an Outbreak of Encephalitis", *Australian J. Exper. Biol. & M. Sc.*, 31: 31.

THELIN, F., and WIRTH, J. (1951), "Epidemic Myalgia (Bornholm Disease). Isolation of a Virus during a Recent Epidemic", *Rev. méd. Suisse Rom.*, 71: 44.

Reports of Cases.

IDIOPATHIC HYPOPARATHYROIDISM.

By FELIX ARDEN,
Brisbane.

Case History.

IN August, 1951, a thin, alert, sandy-haired boy, who was then thirteen years old, had a curious attack while playing football. Of a sudden his hands became stiff and his fingers felt tight, while at the same time his face stiffened and he had a peculiar feeling in the legs. There was no particular pain. He neither fell nor lost consciousness. In a few seconds the attack was over and he went on with the game.

From that time similar attacks followed, at first every week or two and then with increasing frequency, never when he was in bed and hardly ever when he was alone and quiet. They usually came when he was suddenly jolted into activity, as when asked a question unexpectedly in class,

or when a catch came his way while fielding in the slips. They were certainly more frequent at school, almost disappearing during the long holidays, and coming back again at the resumption of term. Within twelve months of the onset he was enduring a minimum of two or three every day and often many more.

Being sensitive about his condition, the lad concealed it from his parents as long as he could, so that it was two months before the family doctor was consulted. Not unnaturally, confronted with a highly-strung adolescent, the doctor made a diagnosis of "nerves" and prescribed phenobarbital.

After a considerable time, when there was no response to treatment but the condition rather grew worse, the boy visited a psychiatrist. This was in March, 1952, seven months after the onset of the trouble. The psychiatrist decided that the only course was to "take him home and belt it out of him" and conveyed these sentiments to the mother. She, with the instinctive wisdom of her kind, consulted another doctor, who had an estimation of the serum calcium content made and, on finding it low, made a diagnosis of tetany. This was in May, 1952. For the next three months the patient consumed large quantities of calcium without any improvement at all.

When seen in August, 1952, he had become rather despondent. The attacks were very frequent, from two to twenty a day, and tended to come on whenever he made a sudden move or changed his position. Although brief, they were quite incapacitating, tying him up temporarily and making him feel foolish. He felt that he was a source of amusement to the other boys in the form, as, of course, he was, and he could not play games. His mother complained that he had an inferiority complex and was irritable at home. At night, even with sleeping tablets, he was most restless and often got up and walked in his sleep. In spite of his troubles he ate well and was growing fast.

Examination revealed him to be a tall, slim boy with a nervous, apologetic manner and quick movements. He spoke rapidly with a slight stammer. His bones and teeth were well developed. Hair, nails and skin were normal. There was no clinical evidence of rickets. The reflexes were brisk and Chvostek's sign was elicited. No other abnormality was found on examination. He had an attack of tetany at his first visit, lasting perhaps ten seconds and chiefly affecting the hands, which assumed the characteristic *accoucheur* position. There was temporary immobility of the face, but not of the eyes. The boy himself complained of a stiff feeling in his hands, arms and cheeks.

X-ray examination of the long bones revealed no evidence of metabolic or nutritional disorder. The laboratory reported a low serum calcium content of 7·8 milligrammes per 100 millilitres (normal 9 to 11 milligrammes), raised alkaline phosphatase content of 24 King-Armstrong units (normal 10 to 20 units), low serum phosphorus content of 2·2 milligrammes per 100 millilitres (normal 4·5 to 5·5 milligrammes). Of this last result more anon. The serum protein content and plasma carbon dioxide content were normal (6·8 grammes per 100 millilitres and 60 volumes *per centum* respectively).

Confirmatory tests at another laboratory showed the serum calcium content to be 8·0 milligrammes and the phosphorus content 7·0 milligrammes per 100 millilitres. The contrast between this phosphorus estimation and the 2·2 milligrammes reported earlier was rather startling.

What one must regard as inadequate treatment was at first prescribed, consisting of 1/10 grain desiccated parathyroid gland twice daily, phenobarbital half a grain in the morning and one grain at night, and a tablet containing 15 grains of dicalcium phosphate and 500 international units of calciferol after each meal. This went on for two months, but he was really no better, remaining only fairly well during the school holidays and having his attacks as usual the following term.

When the boy was seen again in October he was still having attacks, especially when excited, although he was not missing any school and was turning out hopefully to cricket, which he loved. He had another spasm in my rooms.

Testing again showed a low serum calcium content (7·0 milligrammes), a high phosphatase content (30 units), and the serum phosphorus content to be at the relatively high figure of 8·0 milligrammes per 100 millilitres.

Meanwhile the dosage of parathyroid had been increased to 1/10 grain three times daily, the calcium preparation changed to the lactate, the vitamin *D* intake raised to 11,500 units daily and the phenobarbital continued. This alteration had no effect on his attacks, which persisted as before.

A dramatic change occurred about the middle of November when, after some inquiries, high-potency "Ostelin" tablets were ordered and the lad began to take 150,000 units of calciferol each day. Within a week his attacks of tetany ceased completely and his subjective feelings of tension and uneasiness began to disappear. When he reported in December he felt incomparably better and had weathered his school examinations without incident. He still kept his nervous manner and rapid speech, but he had no tetany, no Chvostek sign and more leisurely movements. He looked well and examination revealed no abnormality.

After a month of this treatment his serum calcium level had risen to 9·3 milligrammes per 100 millilitres and the phosphorus level had fallen to 6·0 milligrammes per 100 millilitres. His urine showed a faint cloud of calcium salts with Sulkowitch reagent—a normal reaction.

Since November the boy has remained well. The high intake of calciferol has been maintained and all other therapy gradually discontinued. His calcium tablets were stopped in December and the parathyroid in January. The phenobarbital was reduced to half a grain, at night only, in January, and discontinued in March.

In spite of this he has had no attacks of tetany at all. He says he feels well and looks it. He has concentrated sufficiently on his cricket to win a place in the school team as a spin bowler. His parents say that, although still excitable, he has become easier to live with. They have noticed that he no longer bites his nails so much, that he sleeps more peacefully at night and has completely given up his somnambulism.

He is now being maintained on 100,000 to 150,000 international units of calciferol daily, depending upon the serum levels and the results of the Sulkowitch test, with no calcium intake beyond what is contained in his diet.

The most recent tests show the serum calcium content to be 10 milligrammes per 100 millilitres, the phosphorus content to be 5·5 milligrammes per 100 millilitres, and the alkaline phosphatase content to be 10·9 King-Armstrong units. These findings are within normal limits.

My colleague, Dr. A. Harrison, reports that slit-lamp examination shows no lens opacities. Skull X-ray pictures, for which I am indebted to Dr. H. Masel, reveal patchy cerebral calcification which is more or less symmetrical and situated in the region of the basal ganglia.

Comment.

There are good reasons for believing that the boy whose case history has been described is suffering from hypoparathyroidism, designated "idiopathic" to distinguish it from the variety following accidental removal of the glands.

The condition is rare and, so far as I am aware, no Australian example has been reported. Steinberg and Waldron (1952), who described a case last year, found only 51 others in the world's literature capable of fulfilling the following diagnostic criteria: (*a*) symptoms and signs of chronic tetany, (*b*) low serum calcium content, (*c*) high serum inorganic phosphorus content, (*d*) no evidence of renal insufficiency, (*e*) no evidence of rickets, (*f*) no evidence of chronic intestinal disease, (*g*) no history of thyroid surgery.

Three-fourths of the cases have their origin before the age of sixteen years. There is no significant sex difference.

The great majority of the patients have had previous good health. Little is known of the pathology. The only three post-mortem examinations performed have shown the parathyroids to be either absent or replaced by non-glandular tissue. We appear to be dealing with simple failure of the parathyroid glands, of unknown causation. The symptoms are reasonably constant. Every patient except one has presented himself with frank tetany, convulsions or "tetanic equivalents"—that is, paræsthesia, numbness, muscle cramps or laryngeal stridor. The one exception was a patient whose only symptoms were photophobia and blepharospasm and in whom Chvostek and Trousseau signs were elicited.

In view of the spasms, mistaken diagnoses of epilepsy have been made. Other patients have had genuine epileptic attacks. Diminution in the number and severity of their seizures and improvement in their electroencephalograms have followed the restoration to normal of the serum calcium and phosphorus levels. Other writers have described how the attacks of carpo-pedal spasm tend to be brought about by activity or excitement.

Personality changes have been reported in many instances. This boy, who was at one time very irritable, became "easier to live with" when his condition was controlled. It is humbling to reflect how much our characters can be influenced by the concentration in the serum of inorganic ions.

About half of all the patients whose eyes are examined by slit-lamp have shown lens opacities. Regression of such lens opacities occasionally follows adequate therapy, but it usually persists. One-third of the patients, especially those whose disease started in childhood, have shown defective formation of enamel and dentine and at times retarded eruption of teeth. Only about one-quarter of all patients have dry skin, sparse hair or brittle nails.

The laboratory findings are of interest. Hypocalcæmia is, of course, a constant feature. The lowest recorded levels prior to therapy vary from 3·6 to 8·6 milligrammes of calcium per 100 millilitres, in this patient 7·0 milligrammes. Low urinary excretion of calcium accompanies the low serum level and the patients usually remain in balance although their absorption is poor. Jordan and Kelsall's (1951) patient showed an average daily calcium retention of 28 milligrammes before treatment, but after five weeks the average daily retention rose to 733 milligrammes. This suggests that for a while at least there may be some sort of calcium debt to be repaid to the depots.

High serum phosphorus levels are also constant. The highest recorded readings ranged from 5·2 to 13·3 milligrammes *per centum*; in this patient it was 8·0 milligrammes. Results of phosphorus balance experiments have been variable and are hard to interpret.

The alkaline phosphatase content was normal in most of the cases in which estimations were made. It was, however, distinctly high in Jordan and Kelsall's patient, varying from to to 33 King-Armstrong units, and in mine before treatment (24 to 30 units). I do not understand the reason for this.

Cerebral calcification has been mentioned. Seven out of 22 patients investigated by X rays have shown deposits in the region of the basal ganglia similar to the plates demonstrated here. Identical calcification has been reported in surgical hypoparathyroidism (Medill, 1951). This condition is not reversible, but it does not of itself seem to produce any symptoms. Concerning prognosis, it can be said that with adequate treatment the risk to life is small. Only five of the 52 patients were dead at the time of Steinberg and Waldron's inquiries last year, and three of these had died of unrelated causes. On the other hand, there is no likelihood of "recovery" in the sense of not needing treatment.

The obvious therapeutic aim is to make good the lack of parathyroid hormone. To use parathyroid extract, while logical enough, is not the best method, for the substance is expensive and tolerance eventually develops. Parathyroid transplants, tried in three cases, have been unsuccessful. Fortunately, two other preparations, calciferol and dihydrotachysterol (better known as "A.T.10"), effectively imitate the dual action of the parathyroid in that they increase calcium absorption from the bowel and phosphorus excretion from the kidney.

There is some controversy as to which is the more useful drug.

Of 14 patients treated with calciferol in doses ranging from 30,000 to 400,000 international units daily the results were good or fair in nine cases and poor in five. "A.T.10" was developed specifically for post-thyroidectomy tetany and so far 19 patients with the idiopathic condition have been treated with good results in every instance. The usual dose is three mils of the solution (that is, 3·75 milligrammes) daily until the body chemistry is normal, followed by a maintenance dose of one to seven mils weekly. However, as the cost of treatment with "A.T.10" is higher than with calciferol, it seems reasonable to start with the latter drug, changing to "A.T.10" if necessary.

Calcium therapy by itself does not alter the serum calcium level and has even less chance of affecting the serum phosphorus content. There does not seem to be much object in prescribing extra calcium salts with calciferol or "A.T.10" so long as the diet is adequate.

In similar fashion to the diabetic the patient artificially supplied with parathyroid substitute treads a narrow path and must be protected from the dangers of hypercalcæmia and hypocalcæmia. Frequent estimations of the serum calcium content are necessary, while the response of the urine to the Sulkowitch reagent is a useful rough test. A white cloud indicates excess calcium excretion and a high serum level; no precipitate at all means hypocalcæmia. A faint milkiness is the desired reaction. The patient can be taught to make this test daily and to record his results. With these precautions he can lead a useful and happy life, plagued only by the physician who may become more interested in his phosphorus than in his feelings, in his calcium than in his career.

References.

JORDAN, A., and KELSALL, A. R. (1951), "Observations on a Case of Idiopathic Hypoparathyroidism", *Arch. Int. Med.*, 87: 242.

MEDILL, E. V. (1951), "Bilateral Symmetrical Calcification of the Basal Ganglia Associated with Paratnyroid Insufficiency", *Brit. J. Radiol.*, 24: 685.

STEINBERG, H., and WALDRON, B. R. (1952), "Idiopathic Hypoparathyroidism: An Analysis of Fifty-two Cases", *Medicine*, 31: 133.

Reviews.

Infant Feeding. By Vernon L. Collins, M.D. (Melbourne), M.R.C.P. (London), D.C.H. (London); 1953. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 7" x 4½", pp. 92. Price: 6s.

THE new edition of Dr. Vernon Collins's useful little book has much to recommend it. It is primarily for medical students, but contains all the theoretical knowledge of infant feeding that a general practitioner need have, and much practical information. The book is attractive, is printed on good paper in clear type, and is a very convenient size for frequent reference. The introductory chapter dealing with the normal infant and its progress and requirements follows the modern trend, very happily combining science and common sense to introduce the baby gradually to a routine. Breast feeding, artificial feeding and mixed feeding have a chapter each, and there is a final very helpful chapter dealing with feeding in the abnormal infant, for example, in cœliac disease; this includes a section on the differential diagnosis of vomiting, constipation, crying *et cetera*. Welcome reference is made to psychological maladjustment in the infant, in particular the "hypertonic infant" and its treatment.

It is a pity that such a useful and comprehensive and up-to-date little book has several flaws. The section on breast feeding has failed to keep abreast with modern teaching, engorgement of the breast is still attributed to colostrum blockage of ducts, and no mention is made of the use of œstrogens in its control. Sore nipples are dealt

with unsatisfactorily with no mention of prevention, and flow is still used synonymously with "quantity", and "weaning" with mixed feeding.

It is a sad reflection that no two States in Australia or even pædiatricians for that matter can agree on the details of milk mixtures, and though there is no doubting that Dr. Collins's milk mixtures will be very satisfactory in practice, they are not those in use by the New South Wales baby health centres, for example. We are glad to see that Dr. Collins has not been swept away by the "high protein wave" and still advocates that a "2 part milk, 1 part water mixture" should be introduced at three to four months, not earlier; it does seem a pity, however, to give up the use of cod liver oil emulsion that so conveniently adds naturally occurring vitamins and Calories in favour of concentrates. Many babies need a 20 Calories to the ounce feeding and are unable to take three ounces per pound body weight per day required in lower Calorie feeds.

This book deserves to maintain its popularity, and with some revision of the breast feeding chapter and more consideration of the health department variations in each State, it could fulfil all the requirements of the Australian general practitioner in this field.

Physiologic Therapy for Obstructive Vascular Disease. By Isaac Starr, M.D.; 1953. New York: Grune and Stratton. 9" x 6", pp. 46, with four text figures. Price: $2.50.

THE material for this monograph was first prepared as a George E. Brown Memorial Lecture and was presented before the American Heart Association in April, 1952. The subject matter presents the author's personal experience and opinions on the subject and he does not present the material in the usual review form. In fact, it is refreshing to see a list of only fifty-six references. The whole monograph is presented in essay form and is very easy and delightful to read. It does not cover all aspects of the subject, but deals well and critically with those aspects in which the author has had extensive experience. The most modern surgical approach to some forms of this disease are not mentioned.

The author commences with a description of the treatment of obstructive vascular disease in the years before 1928. Radical amputation was almost invariably used early. Dr. Starr then describes his work with Sir Thomas Lewis and particularly the cutaneous histamine reaction, and pays great tribute to Lewis for initiating a more rational and physiological approach to a subject which had previously been treated entirely on anatomical and pathological principles.

He considers his experiences with various physiological methods of treatment which have been used, many of them now discarded and in some cases dangerous. The various pumping devices appear to help, but in most cases do not justify the expense. Radiant heat had a vogue, but was quickly found to accelerate gangrene. Vasodilators which do not cause a fall in blood pressure or increased cardiac output, such as "Mecholyl" and "Doryl", have often increased blood flow and reduced pain; however, the perfect drug has not yet been discovered. Dr. Starr states quite dogmatically that people suffering from this disease should abstain from smoking and that such abstinence is usually followed by improvement. Sympathectomy usually produces benefit, but in his experience this has been limited to a few years. There seems to be good reason for keeping an acutely diseased foot below heart level, but in the more chronic cases the patient's judgement seems to dictate the best position.

Regarding the newer methods, Dr. Starr considers that introduction of the antibiotics has been the most useful advance and that anticoagulants have not done very much in cases of embolism and thrombosis which did not occur spontaneously before their introduction.

Whilst we cannot recommend this book as an up-to-date text-book on the subject of peripheral vascular disease, it is a book to be read by those interested in the history and philosophical aspects of medicine.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"The Expectant Mother: A Practical Handbook of Advice to the Expectant Mother and to the Newly Married", written by a group of doctors and published under the auspices of the New Zealand Obstetrical and Gynæcological Society, with a foreword by Sir J. Bernard Dawson, K.B.E., Emeritus Professor of Obstetrics and Gynæcology, University of Otago; 1953. Christchurch: Whitcombe and Tombs, Limited. 7¼" x 5", pp. 132. Price: 6s. 6d.

The whole object of this book is to inspire confidence and dispel fear.

"Sex, Society and the Individual: Selected Papers, Revised and Brought Up to Date, from *Marriage Hygiene* (1934-1937) and *The International Journal of Sexology* (1947-1952)", edited by A. P. Pillay, O.B.E., M.B., B.S. (Bombay), and Albert Ellis, Ph.D. (New York); 1953. Bombay: The International Journal of Sexology. Sydney: Angus and Robertson, Limited. 9" x 6", pp. 458, with 28 illustrations. Price: 56s.

The title of the book is an indication of its scope.

"Social Aspects of Disease", by A. Leslie Banks, M.A., M.D., F.R.C.P., D.P.H.; 1953. London: Edward Arnold and Company. 9" x 6", pp. 380. Price: 20s.

Intended primarily for ancillary workers in medical services, but also for medical students and practitioners.

"Psychiatric Treatment: Proceedings of the Association for Research in Nervous and Mental Disease, December 14 and 15, 1951, New York", edited by S. Bernard Wortis, M.D., Morris Herman, M.D., and Clarence C. Hare, M.D.; Volume XXXI; 1953. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9" x 6¼", pp. 466, with 49 illustrations. Price: £4 16s. 9d.

Comprises twenty-four contributions by different authors.

"Textbook of Virology: For Students and Practitioners of Medicine", by A. J. Rhodes, M.D., F.R.C.P. (Edinburgh), and C. E. van Rooyen, M.D., D.Sc. (Edinburgh), M.R.C.P. (London); Second Edition; 1953. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9" x 6¼", pp. 576, with 76 illustrations. Price: £4 6s.

The aim is to present the essential features of the virus and rickettsial diseases of man.

"The Work of WHO, 1952: Annual Report of the Director-General to the World Health Assembly and to the United Nations." Official Records of the World Health Organization, No. 45; 1953. Geneva: World Health Organization. Australia: H. A. Goddard, Proprietary, Limited. 255A George Street, Sydney. 11" x 8½", pp. 214, with 17 plates and 11 maps. Price: 9s.

This report is important because it is the last which will be issued by Dr. Brock Chisholm, who is retiring from the position of Director-General.

"Lind's Treatise on Scurvy: A Bicentenary Volume Containing a Reprint of the First Edition of A Treatise of the Scurvy by James Lind, M.D., with Additional Notes", edited by C. P. Stewart, Ph.D., D.Sc., and Douglas Guthrie, M.D., F.R.C.S.E.; 1953. Edinburgh: The University Press. 9" x 6", pp. 452, with four illustrations. Price: 45s.

This is a reprint of Lind's original work in modern typography with additional biographical notes and notes on the developments up to the present day.

"The Basis of Clinical Neurology: The Anatomy and Physiology of the Nervous System in Their Application to Clinical Neurology", by Samuel Brock, M.D.; Third Edition; 1953. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9½" x 6½", pp. 522, with 124 illustrations. Price: £3 15s. 3d.

The author has attempted "to present neuroanatomy and neurophysiology mainly from the standpoint of clinical usefulness".

"Recurrent Dislocation of the Shoulder", by James A. Dickson, M.D., Alfred W. Humphries, M.D., and Harry W. O'Dell, M.D.; 1953. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9½" x 6", pp. 164, with 78 illustrations. Price: £2 8s. 6d.

The book is planned "to facilitate a clearer understanding of the factors involved in recurrent dislocation of the shoulder".

"The Principles of Neurological Surgery", by Loyal Davis, M.S., M.D., Ph.D., D.Sc. (Hon.); Fourth Edition; 1953. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 9½" x 6½", pp. 544, with 359 illustrations, four in colour. Price: 91s. 6d.

The author does not pretend that this is an exhaustive treatise on the subject; his object is to help the student of medicine to obtain a "better concept of neurological surgery".

The Medical Journal of Australia

SATURDAY, AUGUST 8, 1953.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

PÆDIATRICS.

PRACTICALLY the whole of the present issue of this journal is devoted to pædiatrics, and in particular to the papers presented and the discussions which took place at the annual meeting of the Australian Pædiatric Association, held at Canberra last April. The occasion offers an opportunity for a discussion on the content and scope of pædiatrics, which, to say the least of it, is a fascinating and unique specialty. It has been said with truth that pædiatrics is unique in that it deals with human beings of a special age group. This is true, of course, but it is not the whole truth, for pædiatrics spreads out in many directions. W. S. Craig in a Honyman Gillespie Lecture delivered in June, 1952,[1] showed that pædiatric history was inseparable from changing social events and described the course of the specialty as being amœboid—he entitled his address "Pædiatric Pseudopodia". This was a happy title. We all know that the pseudopodia of an amœba continually change the amœba's form and thus its direction. If pædiatrics was concerned only with the illnesses of children it could not be compared to a living amœba. It is its pseudopodia which give it life and interest, and some of them were brought to light at the Australian Pædiatric Association's meeting.

The papers and discussions published in this issue show that there is ample reason for regarding pædiatrics as a specialty. It is difficult to find evidence of the extent to which general practitioners and pædiatricians care respectively for the children of the community. The only statement available is in the introduction by Waldo E. Nelson to the "Textbook of Pediatrics" edited by him. He refers to a survey of medical care for children sponsored by the American Academy of Pediatrics in cooperation with the United States Children's Bureau and the United States Public Health Services in which it was found that the general practitioner was providing about 75% of the medical care of children and that about one-third of the general practitioner's time was devoted to pædiatrics. What the figures would be for Australia we can only guess. It is perhaps of interest at this point to recall what was known as the Hengrove Experiment, carried out in England and discussed in these columns in July, 1952. This covered a "pædiatric general practice" and was reported by Dr. Norman J. Cook. Our conclusion on that occasion was to question the workability of the proposition. The opinion was expressed that the family doctor (the general practitioner) had the child in his care as a member of a family, subject to the same hazards and stresses as all the members of the family, and was therefore best qualified to look after the child—he could obtain expert help when he needed it. We can thus claim that the papers and discussions of the Australian Pædiatric Society are, or should be, of interest to general practitioner readers of this journal as well as to pædiatricians who did not go to Canberra. The general practitioner, though he may not realize it, is continually making contact with, or coming into close proximity to, the "pseudopodia" of pædiatrics. Let us consider some of these pseudopodia.

The first fact to be remembered about pædiatrics before we think of its pseudopodia or ramifications is that it is concerned with subjects who are in a continuing state of growth and development. Nelson refers to this and expresses the view that it sets pædiatrics apart from other branches of medicine. The growth and development of the child are features of the mind as well as of the body. We know, from observation of children in our family circles, if in no other way, that the environment of a growing child is of the utmost importance to its future well-being, especially in matters of the mind. Medical practitioners have known this for many decades and have paid attention to it in their care of children. The social legislation in the nineteenth century which prevented the exploitation of child labour was one great factor in the promotion of child welfare, and the establishment of organizations dealing with destitute and sick children led to extension of child care in many directions. We need not attempt to trace or even to enumerate these; it must suffice to remind ourselves that the welfare of the child is recognized nowadays as being a matter in which the State is vitally concerned. Thus we have the whole range of preventive medicine in its many facets as one of the main pseudopodia of pædiatrics. It is interesting to note that Craig in his address attaches great importance to what he calls the proper appreciation of the pædiatric point of view. There is, he states, a tendency to attribute the present-day interest of pædiatricians in environmental factors to the compelling influence of "*modern* social medicine"—and at times even to expediency. This "interpretation" he regards as "fundamentally wrong". In the next sentence he writes: "History bears ample testimony to the frequency with which advances in pædiatrics are to be traced to sociological studies of one kind or another." We need not concern ourselves over much with the point which Craig wishes to make. What does matter is that preventive medicine must be recognized always as part, *inter alia*, of

[1] *Edinburgh M. J.*, January, 1953.

pædiatrics and reciprocally pædiatrics as having a formal place in preventive medicine. In the last few years pædiatrics has been compelled to put out a far-reaching pseudopodium. The observations of Gregg on the occurrence of cataract in the children of mothers who suffered from rubella during pregnancy and the extension of this work by him and others have made it necessary for the pædiatrician to take a vital interest in the health of the pregnant woman lest her offspring display some abnormal development which will cripple his whole future. This, by the way, is a pseudopodium which Craig does not emphasize. A moment's thought will show also that the pædiatrician must be seriously concerned in the nutrition of the pregnant woman.

Clearly the scope of pædiatrics is wide and is extending every day. Both authors mentioned insist on this. Nelson sums up a statement on the different views held on the subject with the comment that "concern for the child must antedate conception and extend through the final phases of growth in the period of adolescence". Craig states that pædiatrics should preserve and strengthen its ties with general medicine—general medicine being interpreted to include social and preventive considerations. He does not seem to be quite certain where pædiatrics is going. In one place he writes (and he uses italics to emphasize his view) that although the demands of scientific progress may know no limit, "*events can be relied upon to ensure that in the course of time the practice of pædiatrics as a human art and the practice of pædiatrics as an applied science will be seen in their proper relation the one to the other*". Apparently he dislikes bureaucracy, for he states that he has had some personal practical knowledge of administrative methods in medicine and in industry, and he has no hesitation in saying (again in italics) that "*if professional advice does not receive the attention it merits the time will come when the humanity of hospital care of children will give place to the materialism of factory management*". Looked at carefully, these statements will be seen not to be contradictory but to reveal the fear of the man who made them. It is probably right to say that pædiatrics requires a human approach more than any other specialty. Anything approaching "factory management" in pædiatric practice will be disastrous and make its success impossible. The pædiatrician must be attracted by children as people ("Even children are people.") and must be attractive to them. A skilled pædiatrician can sum up the qualities of a child almost by instinct (or so it would seem), and the way in which children respond to him is often the wonder and the envy of lesser folk. We have set out in some measure the extent of the field of pædiatrics and cannot but agree with Craig that pædiatrics should preserve and strengthen its ties with modern medicine. A good case could be made out for the view that pædiatrics from some points of view is the basis, the foundation, on which general medicine may be regarded as having built its edifice, though unconsciously. While we must leave that argument to others, we can suggest that attention should be paid to the way in which pædiatrics is presented to undergraduate students in medicine so that they may gain a proper understanding of its range and purpose. But that must be the subject of a future discussion.

Current Comment.

MIGRAINE IN CHILDREN.

MIGRAINE is not a common disease of childhood, but George R. Krupp and Arnold P. Friedman[1] have a headache clinic at the Montefiore Hospital for Chronic Diseases in New York, and so they meet with the condition more often than most doctors. In an effort to evaluate the syndrome, Krupp and Friedman have made a special study of 50 children with migraine. By migraine they mean "that form of recurrent headache which is characteristically unilateral, occurs in bouts usually associated with gastro-intestinal and/or ocular symptoms against a background of relative well-being, and is often preceded by visual or psychological disturbances and followed by sleep". The frequent occurrence of headaches in the parents or other members of the family is also mentioned, and it is interesting to note that the study included an interview by a psychiatrist and a social worker of the mothers of the children aimed at assessing the mothers from a psychiatric point of view. One outcome of this is a suggestion that a familial incidence of migraine may in part be due to the factor of exposure to the atmosphere created by the parent's suffering from headaches, rather than to heredity. The children studied were usually of superior intelligence, but all types of personality were observed. The commonest personality traits were sensitivity, cleanliness, thoroughness, the need for approval and the need to take seriously such responsibilities as school and dress. Generally the performance of these children was good, but in some cases headaches were related to unexpressed hostility towards figures of authority whose approval was urgently needed. Other minor psychogenic disturbances, such as nail-biting, occurred in nearly every case. Precipitating factors for attacks could not always be determined, but the usual exciting cause was a stress situation associated with failure, disappointment, rejection, unusual stimulation and anxiety. One of the most common incidents was a family argument, with the child in conflict with its parents or siblings. Examination of the symptomatic picture revealed no significant differences between the migraine attacks of children and those of adults. The condition appeared, however, to be milder in children with a greater preponderance of gastro-intestinal symptoms associated with the headache or "migraine equivalents". For treatment, which must be described as essentially symptomatic, fairly good results were obtained in most cases with one milligramme of ergotamine tartrate and 100 milligrammes of caffeine, provided that it was given early. Krupp and Friedman point out that the effective dosage can be determined by giving one tablet at first and repeating it, if necessary, every half-hour for about four doses; subsequently, the total number of tablets required can be taken as a single dose. Belladonna alkaloids help to reduce side-effects. The other common drug was aspirin, but it was effective in little more than a quarter of the cases. Two important points stressed by Krupp and Friedman are the need to give drug treatment early in an attack if it is to be effective and the fact that treatment must be adapted to the individual patient. The second of these points is even more important in relation to prophylaxis, which is stated to depend upon an understanding of migraine as a total reaction of the child to his environment. Krupp and Friedman consider that parents can usually be made to understand that migraine is closely related to emotional upsets because of what has been observed in their children and in themselves, and because many of them suffer similar headaches. However, while limitation of the patient's activities and rearrangement of schedules to provide regular sleep and regular meals may reduce the frequency of headaches, such restrictions may create new personality and adjustment problems for parent and child. Hence individual handling of the problem is necessary on the doctor's part, preferably with the aid of

[1] *Am. J. Dis. Child.*, February, 1953.

a psychiatric case worker. The experience in this group of 50 cases was that psychiatric investigation of the child and education of the parents on the nature of the disease often produced almost immediate improvement in the child's condition. Krupp and Friedman's discussion of this aspect of the subject is thoughtful and practical in its approach; it would repay reading in the original article.

POLIOMYELITIS IN FRENCH OCEANIA.

DURING the months of March, April and May, 1951, 128 cases of paralytic poliomyelitis, eight of them fatal, were reported from the scattered islands comprising French Oceania. The disease was first diagnosed on Tahiti, the administrative centre and the most heavily populated island in the territory, and it soon became apparent that the outbreak possessed several unusual features. The distribution of cases by age did not conform to the classical infantile age selection pattern of tropical areas. Certain sectors of Tahiti were more heavily stricken than others, and the differences could not be accounted for by variations in the completeness of reporting of cases. Finally, it became apparent that the island had experienced a high paralytic attack rate (360 per 100,000), which was higher than that prevailing during the usual epidemics in the United States, Europe and Australia. Some epidemiological observations on the outbreak have now been published by Leon Rosen and George Thooris,[1] together with notes on the incidence of paralysis following intramuscular injections. They state that although cases of poliomyelitis had been reported on neighbouring islands, none had been previously reported from French Oceania, and it was thought that none had occurred there. However, during the careful survey made during the epidemic, it was found that four people had residual paralysis, which was almost certainly due to poliomyelitis; it dated from 1947 in two cases and from 1950 in the other two cases. Thus, it is very likely that the outbreak which began in March, 1951, was not the first introduction of the disease to Tahiti. The outbreak was explosive in character, 93 of the 109 cases occurring during the five-week period in March and April; eight of them terminated fatally. The attack rate was highest in the age groups ten to fourteen years and fifteen to nineteen years. In some districts the attack rate was higher than in others; when a search was made for a factor to account for this, it was realized that the Taravao Medical Post had been conducting an energetic campaign against treponematosis and a large number of patients had been treated with weekly intramuscular injections of various preparations of arsenic, bismuth and mercury. The site used for the injection was the buttock, alternate sides being used each week. The treatment had been suspended during a measles epidemic, but was resumed on the subsidence of the epidemic in the middle of February, 1951. It was found that, of 700 people who had finished their course of injections for treponematosis before February, 1951, only two contracted poliomyelitis; while of 645 who actually received an injection at the beginning of the poliomyelitis epidemic, 40 developed the disease. In addition, it was found that children under fifteen years of age who had received weekly intramuscular injections in the buttocks for treponematosis experienced a much higher attack rate for paralysis of the lower extremities than did an apparently comparable population which had not recently received injections. Rosen and Thooris discuss these results and some of the related difficulties in the interpretation of their significance. They also refer to the suggestion that the increased incidence amongst those who received treatment for treponematosis might be related to that disease, but make the sapient comment that if the disease had any relation to the occurrence of paralysis, there should not be the difference in attack rate which was found in various parts of the island, and the treated patients, presumably, should have had an advantage rather than an increased liability to poliomyelitis. The results are, indeed, largely what one might expect. Since McCloskey, of Melbourne, in 1950 reported his observations on the relationship of onset of paralytic poliomyelitis and site of paralysis to prophylactic injections against pertussis, it has become generally accepted that injections such as those against treponematosis could play a part in the pattern observed in Tahiti.

[1] *Am. J. Hyg.*, March, 1953.

TREATMENT OF WHOOPING-COUGH WITH ANTIBIOTICS.

A REPORT on the treatment of whooping-cough with antibiotics has been issued by the whooping-cough subcommittee of the Antibiotics Clinical Trials (Non-Tuberculous Conditions) Committee of the Medical Research Council.[1] It is based on a controlled investigation of the value of chloramphenicol and aureomycin in the treatment of this disease carried out in 1950 and 1951 in eight infectious diseases hospitals in England, Scotland and Northern Ireland. The findings agree substantially with those reported by North American investigators. The children treated were aged up to five years; 98 were given chloramphenicol, 96 aureomycin and 100 an inert powder (lactose and quinine), in each case twice daily for seven days. For twenty days from the first day of treatment the number and severity of paroxysms and the occurrence of toxic effects and complications were noted. In addition, swabbings for isolation of *Hæmophilus pertussis* were taken on the patient's admission to hospital and at intervals afterwards. The investigators state that when patients who had symptoms for one to twenty-one days before the beginning of treatment were considered together, the benefit to the treated patients, though consistent during the period of observation, was small. If attention had been directed solely to the straightforward comparison of the whole of the treated and control groups, it would have been concluded that the results were disappointing and that chemotherapy failed to produce a clear-cut clinical cure. However, when treatment was instituted within eight days from the onset of the earliest symptoms, the differences in results between the groups were greater, and some of the differences were statistically significant. Even with early cases, however, the effect of the drugs was not dramatic. No difference was detected between the effect of aureomycin and that of chloramphenicol. On the third and eighth days of observation, but not on the first, second and eleventh days of observation, the proportion of patients from whom *H. pertussis* was isolated was greater in the control group than in the treated group.

The subcommittee offers some speculations on the significance of the results in relation to "our scanty knowledge of the pathogenesis of the disease". It is pointed out that in an acute infectious disease it might be expected that elimination of the infecting agent would be followed by rapid disappearance of the signs which that agent initiated. Such an immediate amelioration in the effects of whooping-cough is not observed, and that fact may support a view that infection with *H. pertussis* sets in train a sequence of events which, once it has started, is not greatly affected by elimination of the pathogen. The present study suggests that early recognition and early antibiotic therapy of whooping-cough may help to reduce the severity of the infection, but that little is to be gained from antibiotic treatment of patients who have had even slight symptoms for more than a week. This means that the position is not greatly advanced from that obtaining three and a half years ago when we discussed in these columns the value of streptomycin in the treatment of whooping-cough.[2] The statement of a group of investigators was then quoted that it is difficult to arrive at a true estimate of the effects of any therapeutic agent in a disease like pertussis. The comment made at the time is still valid—that this really amounts to an admission that no agent gives dramatic results.

[1] *Lancet*, June 6, 1953.

[2] M. J. AUSTRALIA, February 18, 1953.

Abstracts from Medical Literature.

PÆDIATRICS.

Prophylaxis of Rheumatic Fever.

K. H. KOHN *et alii* (*J.A.M.A.*, January 31, 1953) discuss the prophylaxis of rheumatic fever with orally administered penicillin. They state that they have reduced the frequency of rheumatic fever recurrences and the number of group A β-hæmolytic streptococci in the throat by oral administration of penicillin over a period of five years in Chicago. Two tablets of 100,000 units of penicillin were given orally four times a day for seven consecutive days, the first school week of every month from October to July. Throat and nose cultures were made before each monthly course of penicillin. Control groups of children who did not receive penicillin were also studied. The results showed very few recurrences in the treated group compared with the untreated groups—1·5% of recurrences were noted in those treated, as compared with 18% to 20% of recurrences in the untreated. Sulphonamide drugs and penicillin throat lozenges are not recommended in this field. The authors advise penicillin prophylaxis for all rheumatic children up to the age of twenty-five years.

Allergic Encephalopathy with Papilloedema.

J. W. DEVANNEY, JUNIOR, AND J. H. SHEA (*Arch. Neurol. & Psychiat.*, December, 1952) report a case in a child of nine years of allergic involvement of the nervous system, a condition which is rarely seen. They suggest that allergy should be considered as a differential diagnosis in the vague group of disturbances classified as obscure encephalopathy or pseudotumour. They give a detailed account of the case in which, among other signs, bilateral papilloedema was found with venous distension and engorgement. The authors consider that these signs are indicative of a generalized increase in intracranial pressure and that papilloedema is more likely due to this than to a localized allergic response in the optic nerves. They consider a diagnosis of allergic encephalopathy tenable after a careful history is taken and a prompt response is noted to antihistamine therapy. However, proceeding in this manner is possible only if the condition has not progressed to a point where air studies are imperative for diagnosis because the patient's life or vision is endangered by increased intracranial pressure.

Supratentorial Tumours in Children.

R. H. MILLER, W. MCK. CRAIG AND J. W. KERNOHAN (*Arch. Neurol. & Psychiat.*, December, 1952) analyse 190 cases of supratentorial tumours in children from birth to fourteen years. Their records were obtained from the Mayo Clinic. Of the 190 tumours, 97 were gliomata, and of these 58 were astrocytomata, 24 ependymomata, 11 oligodendrogliomata, and four neuroastrocytomata. Thirty-five cases of craniopharyngioma and 14 of pinealoma occurred, in that order of frequency, followed by meningiomata, sarcomata, pituitary adenomata, blood vessel tumours, epidermoid tumours and tuberculomata. Sixty-four tumours were located in the third ventricle; the next commonest situation was the parietal lobe, followed by the temporal and frontal lobes. Frequency of occurrence increased with the age of the patients. Malignancy of grades 2 and 3 was the commonest. The most frequently occurring symptoms and signs were headaches, vomiting, visual disturbances and papilloedema; changes in the *sella turcica*, abnormal calcium deposits and separation of cranial sutures were the commonest radiological signs. Of 34 patients with gliomata, 14 had survived operation for five years or more, and of 16 patients with craniopharyngiomata, 11 had survived for five years.

Osteochondritis Dissecans in Children.

W. T. GREEN AND H. H. BANKS (*J. Bone & Joint Surg.*, January, 1953) record their experience with 27 patients affected by *osteochondritis dissecans*. All were children under the age of fifteen years, and the sex incidence was a ratio of three to one favouring the male, which is the proportion ordinarily seen in older patients. There were 36 joints involved—32 knees, three elbows and one ankle. Eight patients had lesions in more than one joint. In most instances the multiple lesions were symmetrical in distribution. The medial femoral condyle was the site most commonly affected; this occurred in 18 patients, 13 of the lesions being unilateral and five bilateral. The lateral femoral condyle was affected in three patients, of whom one had bilateral lesions. The patients with *osteochondritis dissecans*, furthermore, had a surprisingly high incidence of osteochondritis of the ordinary type in other areas. The authors state that whilst the diagnosis of *osteochondritis dissecans* may be suspected from the clinical history and physical findings, it can ordinarily be confirmed only by X-ray examination. The lesions in the child present a crater-like area of rarefaction affecting the articular subchondral bone, just as they do in the adult. Most of the lesions were recognized in the ordinary antero-posterior and lateral projections. The authors believe, however, that postero-anterior tunnel views should be part of the routine X-ray examination. Most satisfactory tunnel view, in their experience, is obtained by placing the patient prone, flexing the knee to 90° and then directing the tube in a caudal direction so that the rays intercept the joint at an angle between 20° and 40°, depending on how far posteriorly in the condyle one wants to emphasize the visualization. Of the 36 joints in this series 25 were treated without surgery. In some cases the knee joint was protected by the use of a Thomas patten-bottom brace, which prevents the joint from bearing weight. In another group a plaster cast was applied with the knee in the amount of flexion which was needed to remove the pressure of weight-bearing from the lesion; this was usually about 30° of flexion. For patients treated by the patten-bottom brace the average period of protection was seven months. Patients who wore plaster cylinders were protected for shorter periods on the average than were those who wore braces. In 18 of the 25 cases in which joints were treated by protection, at least a year has elapsed since the end of treatment. The result has been classified as excellent in 17 cases. Of the seven patients who have not yet been followed for one year after the end of treatment, four have now no defect evident by X-ray examination and are allowed full activity. Three are still protected, with the lesion apparently healing. The authors believe that the results in nine patients who were treated surgically should not be compared with the results in those treated by protection, since theirs were not comparable cases. They conclude that *osteochondritis dissecans* is not uncommon in children, and that when the lesion is protected, the process can heal quite promptly. This can occur even in cases in which apparent sequestrum-formation occurs within the confines of the cavity of the osteochondritic lesion.

Chronic Laryngeal Stenosis in Children.

ROBERTO MACHADO (*Arch. Otolaryng.*, November, 1952) states that in children the laryngeal cartilages are still growing, and if they are properly moulded good laryngeal patency and a good voice will be obtained. For this reason the dilatation method is preferred to surgical procedures for laryngeal stenosis in children. Three children with chronic laryngeal stenosis form the basis of the discussion. The ætiological factors were prolonged intubation, high tracheotomy and over-long sojourn of a tracheal cannula. Progressive continuous dilatation of the laryngeal stenosis was carried out with a series of rubber Nelaton bougies numbers 6 to 20 (French). The first step is to pass a thread through the stenosis under direct laryngoscopy and to bring the thread out through the tracheal stoma. The Nelaton bougie suitably lubricated is drawn into the tracheal stoma by the attached thread and thence upwards through the larynx and pharynx and out through one nasal fossa. One end of the attached thread is fixed to the tracheotomy cannula below, and the other lies loosely through the nostril. Every week the bougie is changed for a larger one, the one already inserted being used as a guide and traction for the next. The bougie which obtains the desired dilatation is changed on three consecutive weekly occasions for another of the same size. After this, decannulation of the trachea is carried out by the regular procedures, and finally the stoma is closed by reconstructive surgery. The procedure resembles the Tucker method of retrograde dilatation of œsophageal strictures. The presence of the indwelling bougie through the larynx, pharynx and nose is well tolerated, and swallowing of food is managed without difficulty.

Streptomycin and Spinal Fluid Sugar Estimations in Tuberculous Meningitis.

ROBERT H. FRIEDMAN *et alii* (*J. Pediat.*, February, 1953) discuss the significance of sugar estimations in the cerebrospinal fluid of children with tuberculous meningitis treated with streptomycin. They point out that streptomycin acts as a glucose-like reducing agent and as little as 0·5 milligramme *per centum* has a "glucose equivalent" of 13·8 milligrammes *per centum* unless precipitation of the non-glucose-reducing substance is first effected with copper sulphate and sodium tungstate. When this is done, the concentration of

streptomycin needs to exceed 5·0 milligrammes *per centum* before it invalidates the true glucose determination. The Hagedorn-Jensen method does not give reliable results in the presence of streptomycin. The authors describe detailed studies in five children. Two had had no previous treatment, and one of these had a cerebro-spinal fluid block. Intrathecal injection of 50 milligrammes of streptomycin caused a rapid rise in concentration of this drug, and then a fall, at first rapid and then slow over twenty-four hours. The cerebro-spinal fluid true glucose level in the first patient showed a rapid rise within two to eight hours, followed by a rapid fall. At the time of this rise, there was a distinct fall in blood sugar level. In the second case a similar rise and fall of streptomycin occurred, but the cerebro-spinal fluid glucose level showed only a slow steady rise of 14 milligrammes *per centum* in twenty-four hours. In the other three cases the children had had long treatment with streptomycin before this test period. In two of them intrathecal streptomycin therapy caused little change in cerebro-spinal fluid glucose level within twenty-four hours of injection. The other patient had had no intrathecal streptomycin therapy for one month, and 50 milligrammes given by this route caused a rapid rise in her cerebro-spinal fluid glucose level within twenty-four hours. A gradual rise in the cerebro-spinal fluid sugar level during the course of treatment did not always correspond with clinical improvement or indicate a successful outcome. The authors suggest that the fall in cerebro-spinal fluid glucose level in meningitis is due to excess oxidation of sugar by the diseased meninges. In normal tissues streptomycin has no effect on the carbohydrate metabolism of cells, but in tuberculous meningitis they suppose that the drug inhibits the overactive glycolysis and so causes a rise in sugar levels.

Wilms Tumours.

M. P. REISER AND C. D. CREEVY (*Urol. Survey*, October, 1952) have made an overall review of Wilms tumours described in the literature, and analyse 23 histologically proved cases in which the patient was treated at their hospital (University of Minnesota). They state that 20% of all neoplasms in infants and young children arise in the kidney, in contrast to 0·5% of those in adults. Wilms tumours are congenital in origin, and nearly all appear before the age of five years. The clinical course is usually silent until a large mass is discovered. They are highly malignant, they result in death if untreated, "dissemination" occurs late, and the histological picture is variable. As regards their origin, if the microscopic appearance resembles that of the kidney, with primitive glomeruli and tubules, the predominating contributing factor is the renal blastema. However, if the tumour *Anlage* separates before the urogenital ridge has formed the nephrotome, the multipotency of the cells may account for the diversity of tissue seen histologically. The term "embryonal nephroma" has been proposed for these tumours. Symptoms, unfortunately, appear late. The classical triad of pain, tumour and hæmaturia, while common in adults, occurs in less than 20% of renal neoplasms in early life. In about 90% of cases the first sign is an adominal mass noticed by the mother or the physician. Excretion urography is the most useful single diagnostic measure. Considerable difference of opinion exists on the treatment of these tumours, particularly as regards the use of irradiation. There is no doubt that most of them are radiosensitive in the sense that they shrink remarkably and rapidly after irradiation, but few are sensitive in the sense that all the cells of a given tumour can be killed. Most surgeons have been inclined to use irradiation both before and after nephrectomy, but some have seen metastases appear during pre-operative irradiation, and prefer to use it post-operatively only. When it does not seem possible to perform nephrectomy, dependence on irradiation alone is obligatory, and in some cases this has achieved moderately good results. About 50% of Wilms tumours are found to be inoperable. During the twenty years from 1930 to 1950, 23 patients with histologically proved Wilms tumour were treated at the University of Minnesota Hospital. Twenty-one were subjected to nephrectomy and irradiation; one was treated by irradiation alone, since pulmonary metastases were present; no treatment was considered advisable in the other cases, since the lesion was bilateral. The results were as follows: four out of seven patients treated five or more years before were living and well for five and one-quarter to eleven and two-thirds years; eight out of 15 patients treated before 1949 were living two or more years later; one out of five patients treated by X-ray irradiation alone was well after nine years.

The Family as a Reservoir of Infection.

C. HENRY KEMPE (*J.A.M.A.*, April 25, 1953) discusses the family as a reservoir of childhood infections. He states that the purpose of his paper is to point out the frequency and nature of a number of bacterial infections rampant in a given family. He places stress on the variety of clinical diagnoses that may be made despite the fact that a single pathogen may be causing all the disease. He states that recognition of family infection and treatment of the entire family, including those who are not ill, with the best antibiotics available ostensibly offers the best hope for the management and prevention of repeated reinfection from the family source of children who are chronically ill.

Cortisone and ACTH for Rheumatic Carditis.

LEO M. TARAN *et alii* (*Am. J. Med.*, March, 1953) report the clinical effects of cortisone and ACTH on protracted rheumatic carditis in 16 rheumatic children, aged seven to sixteen years. They state that all the children showed clinical improvement. Exudative phenomena were readily suppressed during hormone therapy. All the patients continued to show clinical and laboratory evidence of rheumatic carditis when therapy was discontinued. The authors state that cortisone and ACTH seem to "damp" the symptomatology in protracted carditis only during the period when the hormone is administered; interruption of therapy is followed by a return of carditis to its pre-treatment level. None of the patients studied showed regression in clinical evidence of endocarditis or cardiac hypertrophy. Many patients showed untoward effects. Ten patients had one or several signs of Cushing's syndrome; nine had psychological effects, euphoria or depression; three suffered from syncope after parenteral administration of cortisone acetate. The authors consider that it remains to be proven whether this form of therapy significantly affects the course of rheumatic carditis in cases of long standing. Further exploration in dosage schedule and method of administration may provide a more definite answer to this problem.

Intussusception of the Vermiform Appendix in Children.

ISABELLA FORSHALL (*Brit. J. Surg.*, January, 1953) reports seven cases of intussusception of the vermiform appendix in children. Five of the children were male, two were female. The youngest was aged ten months, the eldest eleven years and ten months. The shortest period of symptoms was forty-eight hours, the longest three and a half months. Three patients had been initially under the care of physicians, one with a diagnosis of constipation and another of gastro-enteritis; in the third case no diagnosis had been made. The pre-operative diagnosis was intussusception in six cases and appendicitis in one case. In five cases the invagination of the appendix was incomplete; in two the appendix was turned completely inside out. In six cases secondary caeco-colic intussusception had occurred; in one simple invagination of the appendix had occurred. Pain was the most important symptom in the six older children; in the infant's history pain was not mentioned. Blood was noticed in the stools of the two youngest children. A precipitating condition in the lumen or in the wall of the appendix was not found in any case. A wide appendicular lumen was noted in five and probably six cases. The fœtal type of caecum was noted in two cases.

Birthmarks on Infants.

A. G. PRATT (*Arch. Dermat. & Syph.*, March, 1953) states that he has been unable to find any recorded information on the natural rate of occurrence of the so-called strawberry mark. To determine this he examined 1096 infants (879 white and 217 Negro), most of them being four to eight days old. Strawberry marks were found on 1·1% of the white and 1·4% of the Negro infants; the major type of *nævus flammeus* on 0·5% of the white and none of the Negro infants; the minor type of *nævus flammeus* on 48% of the white and 39% of the Negro infants; macular pigmented nævi on 2·7% of the white and 15·6% of the Negro infants; Mongolian spot on 0·5% of the white and 60% of the Negro infants. A few other less common birthmarks were also seen.

Fœtal Mortality in Cæsarean Section.

H. R. LITCHFIELD *et alii* (*J.A.M.A.*, March 7, 1953) analyse a total of 969 Cæsarean sections carried out over a seven-year period. They consider that, although the fœtal mortality associated with Cæsarean section is quite high, this is the result of the complication of pregnancy necessitating the section, rather than the section *per se*, and that the incidence of death can be reduced through appropriate obstetric and pædiatric measures.

Medical Societies.

AUSTRALIAN PÆDIATRIC ASSOCIATION.

THE annual meeting of the Australian Pædiatric Association was held at the Australian Institute of Anatomy, Canberra, on June 9 to 12, 1953. Dr. Robert Southby, the President, occupied the chair and 39 members were present. The Association entertained as official guests officers of the Commonwealth Department of Health, members of the staff of the Australian National University, and the Editor of THE MEDICAL JOURNAL OF AUSTRALIA. Seventeen private guests attended the meeting.

BUSINESS PROCEEDINGS.

Annual Report and Financial Statement.

The annual report and financial statement for the preceding twelve months were received and adopted.

Obituary.

The President reported with deep regret the deaths of Dr. H. Douglas Stephens and Dr. Norman Cunningham. The meeting approved of the preparation of a special minute recording the services of Dr. H. D. Stephens, the first President of the Association.

Election of Office-Bearers.

The following office-bearers were elected for the year 1953-1954:

President: Dr. P. A. Earnshaw.

Vice-President: Professor Lorimer Dods.

Honorary Secretary and Treasurer: Dr. D. C. Jackson.

Executive Committee: Dr. R. Southby (Past President, Victoria), Dr. R. Wall (Tasmania), Dr. R. Crisp (Western Australia), Dr. M. Cockburn (South Australia).

Election of Members.

The following were elected by ballot to membership of the Association:

Honorary Members: Dr. Helen Mayo (South Australia), Dr. P. L. Hipsley (New South Wales).

Ordinary Members: Dr. N. Barsden (Western Australia), Dr. G. Ch. de Crespigny (South Australia), Dr. D. L. Dey (New South Wales), Dr. Jean S. Isbister (New South Wales), Dr. C. W. G. Lee (New South Wales), Dr. W. B. Macdonald (Victoria), Dr. H. McLorinan (Victoria), Dr. H. C. Murphy (Queensland), Dr. N. Nicholson (Victoria), Dr. H. Rischbieth (South Australia), Dr. S. E. J. Robertson (New South Wales), Dr. E. K. Turner (Victoria), Dr. Clifton Walker (New South Wales).

Standing Committee on Child Health Facilities.

The meeting approved the formation of a standing committee to continue and to coordinate the Association's survey of child health facilities in Australia. The following were subsequently appointed members: Dr. M. T. Cockburn (Secretary), Dr. E. Sims (Executive Officer), Dr. F. W. Clements, Dr. V. L. Collins, Dr. D. C. Jackson, Dr. R. H. Crisp, Dr. R. Wall.

Fourth Annual Meeting.

It was decided that the fourth annual meeting should be held at Canberra in April, 1954.

SCIENTIFIC PROCEEDINGS.

Seminar on Fluid and Electrolyte Problems.

A seminar on fluid and electrolyte problems was held on April 10, 1953, Professor Lorimer Dods in the chair.

Professor Dods said that they proposed to discuss certain fluid and electrolyte problems of pædiatric practice and had chosen for special attention the fluid and electrolyte requirements of the newborn infant, of the child suffering from diarrhœa and of the child with intestinal obstruction. They hoped to discuss these three problems from the point of view of the pædiatrician working in a modern hospital, aided and abetted by an active well-equipped laboratory, and also from the very practical point of view of the country practitioner who had none of these things. The language spoken would be that of milliequivalents. There was no need for him to defend this choice of dialect, as he knew that most of those present would agree that it was only by talking and thinking in these terms of chemical equivalence that they could have any real concept of electrolyte patterns. Without this concept of electrolyte anatomy, any discussion of the problem must necessarily be very vague and nebulous.

Physiological Principles.

Professor Dods then invited Dr. W. B. Macdonald, one of the guest speakers, to define and review some very significant physiological principles which were obviously essential for any adequate understanding of the problems to be discussed.

Dr. W. B. Macdonald (Melbourne) read a paper entitled "Fluid and Electrolyte Problems: Physiological Principles" (see page 197).

The Problem in the Newborn.

Professor Dods then said that Dr. Kate Campbell, with her very wide and extraordinarily sound knowledge of that physiologically immature and slightly œdematous animal, the newborn infant, had agreed to tackle the extraordinarily difficult problem of defining the fluid and salt allowances of this age period. She intended to approach this problem from the very practical point of view of the general practitioner or the obstetrician who had no help from the laboratory.

Dr. Kate Campbell (Melbourne) then read a paper entitled "Fluid and Electrolyte Problems: Problems in the Newborn" (see page 201).

Dr. Charles Lee (Sydney), one of the guests, opening the discussion on the invitation of Professor Dods, said that all would agree that Dr. Campbell had thrown a practical light on a most difficult subject. There were undoubtedly factors other than renal factors involved in maintaining the infant's fluid and electrolyte homœostasis. It was known that the adrenocortical steroids affected water and electrolyte balance both by their apparent primary effect on water and electrolyte shift in the tissues and probably by a variable effect on sodium and chlorine reabsorption in the renal tubules. It had been shown that the production of, or response to, antidiuretic hormone in these infants might be at fault. The net result of these factors and the renal ones discussed by Dr. Campbell seemed to be that the young infant, particularly the premature, had some difficulty in dealing with a solute load and required a greater amount of fluid than the older infant to excrete the same amount of solute. The obvious and important application of this principle to concentrate feedings or the giving of electrolytes had been emphasized by Dr. Campbell.

The subject of œdema in premature infants was, Dr. Lee thought, a most fascinating one. Did they believe that this œdema predisposed to complications, cerebral or respiratory, and should an effort be made to correct it? If so, how? Had potassium a place, as suggested by Smith and Yudkin in their studies on mineral retention in premature infants? One was inclined to believe that this œdema associated with a greater percentage of extracellular fluid was a process of adaptation to the infant's unexpected change of environment and that in the present state of knowledge it was best not interfered with.

Dr. Lee asked Dr. Campbell why she used glucose and fifth normal saline and not glucose and water in her treatment of the severely jaundiced premature infant.

Dr. Kate Campbell said that it was her impression that the babies who showed the more marked œdema were the babies who had been subjected to hyperæmia during delivery, and as such were not in as good condition as the non-œdematous infant. One would expect that such infants would be more susceptible to infection.

Dr. F. Arden (Brisbane) asked why delayed feeding had become so popular.

Dr. Campbell replied that although delayed feeding had originally been introduced in an attempt to "dry out" the baby, its main benefit was that it prevented the vomiting and aspiration of feedings. As indicated previously, it was not possible to rid the baby of water without ridding it of electrolytes, so that the original idea of "drying out" the baby was unphysiological.

Dr. J. Colebatch (Melbourne) asked, with regard to unexplained œdema of the newborn, how a man in practice without electrolyte tests available could tell whether the withholding of fluids was indicated, if ever; also whether Dr. Campbell considered that dehydration fever was ever caused solely by inadequate presentation of fluid.

Dr. Campbell said that it was her practice to feed a baby suffering from unexplained œdema according to his appetite. She thought that played a significant part in producing dehydration of the newborn infant. In "nature nurseries" where babies were kept at the mother's bedside and fed when they were crying so that crying was reduced to a minimum, no babies were given boiled water and none showed dehydration, even at the height of a heat wave. In other words, "crying produced drying".

Dr. M. L. Powell (Melbourne) asked whether Dr. Campbell thought that the excessive use of hot water bottles and also the high temperatures which prevailed in most nurseries had any bearing on dehydration fever.

Dr. Campbell replied that if the temperature of the nursery was high the baby would "dry out" more quickly.

Dr. V. L. Collins (Melbourne) asked why the newborn baby who was dehydrated became febrile. Fever with dehydration was uncommon in older infants. They often saw much more severe states of dehydration in older infants, without any fever. What accounted for this difference?

Dr. Campbell replied that premature babies who were "very dry" did not run a temperature and could go for a long time without passing urine before "running a temperature". She had seen one baby who did not urinate for fifty-two hours and who was not feverish.

Dr. W. B. Macdonald (Melbourne) said that water depletion affected small babies more than older infants, because of their relatively greater surface area and hence greater water loss. Cellular dehydration might be a factor in limiting by some means further skin water loss, thus leading to a rise of body temperature.

Dr. R. Wall (Launceston) asked whether dehydration fever was more common in big babies and if this related to post-maturity. In view of the fact that premature babies particularly were in an acidotic state, he wondered whether it was desirable that one should withhold artificial feedings for longer than was customary and whether a very diluted feeding should be used.

Dr. Campbell replied that a big baby needed relatively more fluid than a small baby. With premature babies, if there was no breast milk she usually gave something like Nestlé's condensed milk starting with a dilution of 1 in 16.

Dr. M. L. Powell asked when the feeding of a newborn infant should be started.

Dr. Campbell replied that babies over three pounds in weight should be fed in twenty-four hours and babies under three pounds should be fed in forty-eight hours.

Dr. Elizabeth Turner (Melbourne) asked Dr. Macdonald whether he considered that hyperbilirubinæmia affected kernicterus in premature babies and whether bilirubin altered the osmolarity of plasma.

Dr. W. B. Macdonald (Melbourne), in reply to the second part of Dr. Turner's question, said that the osmotic effect of bilirubin was very small compared to electrolytes and could account for only minimal water shift from cells.

Dr. M. T. Cockburn (Adelaide) had the impression that delay in introducing fluid by mouth to premature infants was an important factor in the lessened mortality rate amongst those infants. He considered that no premature babies should be given fluids until all visible œdema had disappeared, and this attitude was in accordance with Dr. Clement Smith's teaching.

Entero-Colitis.

Professor Lorimer Dods (Sydney) said that Dr. Howard Williams, whose clinical research unit had been making a special study of the fluid and electrolyte requirements of the infant suffering from acute gastro-enteritis, would discuss this problem both from the point of view of his own department and also from that of the general practitioner who was faced with the management of this condition in a small country hospital.

Dr. Howard Williams (Melbourne) read a paper entitled "The Pathological and Physiological Assessment and Management of 'Dehydration' in Entero-Colitis" (see page 203).

Professor Dods asked Dr. Bryan Dowd, a guest speaker, to open the discussion.

Dr. Bryan Dowd (Sydney) referred to milliequivalents and said that he did not agree that they should all learn to speak this reasonably new language, but if they did, would they not also have to correct certain obvious anomalies which immediately arose in their nomenclature?

"Normal saline", for example, was a term which had always seemed to him somewhat inappropriate. The solution which they so named, of approximately 0·9% sodium chloride, was not, of course, "chemically normal", and was perhaps more aptly called "physiological", but in reality contained about 0·9 gramme of chlorine (as sodium chloride) in each 100 millilitres. What had to be learned now was that it contained some 150 odd milliequivalents of Na^+ and some 150 odd milliequivalents of Cl^- in each litre. Only in this way would they be consistent in their thinking.

The nominal confusion was, however, merely minor when they began to reflect upon the many eponyms which littered the electrolytic jargon. To apply the names of Hartmann and Ringer and Butler to their respective fluids was perhaps historically praiseworthy, but when a man like Darrow compounded a fluid and subsequently modified it in some way over a period of time on more than one occasion and when they began to speak of these solutions as "Darrow over two" and "Darrow over three" or "Darrow 1946" and "Darrow 1947", surely they were only making ludicrous complexities for themselves. As language became simpler so did thinking.

The fundamental solution to the problem was the one which Dr. Williams had demonstrated—to choose a universal language and to speak it logically. For this alone, to say nothing of all the other things which Dr. Williams had said that morning, they should feel most grateful.

Dr. John Beveridge (Sydney) said that he was in the happy position of having grown up with the language of milliequivalents. Consequently it had become necessary for him to learn the language "milligrammes per cent." so that he might understand what others wrote. He was very grateful to Dr. Williams for his address. His ideas were rather similar to those which he (Dr. Beveridge) and others were hoping to achieve in their own gastro-enteritis unit. Dr. Beveridge entirely agreed with Dr. Williams that it was essential to have in charge of such a unit the one physician and also the one registrar who was trained in electrolyte and fluid management.

There was one question which he wished to ask—had Dr. Williams considered the use of whey in the management of these children? Its electrolyte composition seemed to Dr. Beveridge to be very satisfactory and could well replace the polyelectrolyte mixture which Dr. Williams had suggested.

Dr. H. Williams, in reply to Dr. Beveridge's question concerning the use of whey as an electrolyte supplement, said that he had not used whey, but as the larger proportion of the electrolytes was contained in milk there was no reason why it should not be used. Dr. Williams considered that a variety of electrolyte mixtures could give equally good results. The important principle was that sodium and potassium loss from diarrhœa might exceed the sodium and potassium intake if diluted milk feedings were given and it was essential to supplement these feedings with electrolytes. The method used at the Children's Hospital was merely one effective way of tackling the problem.

Dr. D. C. Jackson (Brisbane) said that their practice in Brisbane followed that outlined by Dr. Howard Williams, and they had been teaching their students and "residents" to use half-normal saline as he recommended. However, Dr. Jackson was rather concerned about those who left them to go to places which had not a cooperative dispensary. Half-normal saline was not on the list of pharmaceutical benefits, nor was it put up by the drug houses. Was it safe and advisable for men working alone to make up their own solutions?

Dr. Jackson's second point concerned the use of lactate and potassium-containing solutions in the later stages of maintenance. Until their hospital laboratory could carry out serum potassium estimations he felt it unsafe to attempt potassium replacement. He therefore used Hartmann's solution which contained comparatively little potassium. Hartmann's solution was not on the pharmaceutical benefits list, but Darrow's solution (vintage unstated) which contained much more potassium was on the list. Was it safe for a man working "blind" (without laboratory facilities) to use Darrow's solution as a maintenance infusion for young infants?

Dr. H. Williams, in reply to Dr. Jackson's question about the safety of the intravenous administration of potassium, especially Darrow's solution, without access to serum potassium estimations, said he considered that potassium was quite safe to administer intravenously up to three milliequivalents per litre per kilogram over twenty-four hours. Adequate renal function was essential before potassium administration was commenced. The nursing staff should be carefully instructed to watch the drip and thus ensure an

even administration over the twenty-four hour period. If these simple rules were observed, serum potassium levels were not necessary for the checking of therapy. Darrow's solution contained 40 milliequivalents per litre and was safe to give, provided the total amount of potassium did not exceed three milliequivalents per litre per kilogram per twenty-four hours and was given evenly over this period. It was certainly not safe to administer a solution containing 40 milliequivalents per litre of potassium rapidly. Dr. Williams thought that the safe procedure was to estimate the day's total potassium requirement and then to make up a solution containing this amount and administer it evenly throughout the day.

Dr. R. Southby (Melbourne) thought that probably a good many babies died of dehydration, as Dr. Williams had suggested. Some babies who came to the Children's Hospital when acutely ill probably died of toxæmia. Dr. Southby wondered whether a country practitioner might occasionally use the intraperitoneal route as a method of giving fluid.

Dr. H. Williams, in reply, said that he considered that the amount of water which could be given was so small that it could be of little real use in the severely dehydrated child who could be treated only by an intravenous drip. In mild dehydration he considered that the use of a continuous intragastric drip was sounder treatment than giving an intraperitoneal injection. If the intragastric drip failed to relieve dehydration this intravenous infusion would be necessary.

Dr. Felix Arden (Brisbane) agreed with Dr. Dowd's remarks about nomenclature and with all that previous speakers had said about the necessity for thinking in terms of milliequivalents when dealing with solutions of electrolytes for parenteral use. However, some action was necessary and he urged that the Pædiatric Association approach the Drug Houses of Australia with the request that "Soluvac" solutions be labelled in milliequivalents as well as with their more conventional but outmoded titles.

Dr. W. B. Macdonald, in reply to Dr. Arden, said that potassium phosphate was used at the Melbourne Children's Hospital because as phosphate was such a prominent constituent of cellular substance, it seemed logical to use it as the corresponding anion to potassium. It was immaterial whether the acid or basic phosphate was given when small quantities were being used, as there was an adjustment to a constant ratio according to the pH of plasma, and one preparation was less cumbersome and less confusing to the nursing staff. It was important to reduce the glucose concentration of solutions when giving a concentrated electrolyte solution, because the kidney had to do extra work and use extra water to excrete any excess. A 5% solution of glucose, even at the infusion rate of one ounce per hour, provided the infant with 35 grammes of glucose per day which was well above the maximum protein and sodium sparing effects and the amount needed to prevent acidosis. If one was using concentrated solutions it was advisable to reduce the glucose solution to 2·5%, and this had recently been done at the Children's Hospital, Melbourne.

In reference to Dr. Dowd's comments, Dr. Macdonald said that a chemically normal sodium chloride solution was over six times as concentrated as a so-called physiologically normal solution, which was physiological only in its concentration.

Intestinal Obstruction.

Professor Lorimer Dods said that Dr. E. S. Stuckey had been asked to review the problem of intravenous fluid therapy in relation to intestinal obstruction. As that was a very tall order, Dr. Stuckey would limit his remarks to the problems of jejunal or upper ileal obstruction, in other words, he would not discuss in any detail the colonic type of obstruction or obstructions above the ampulla of Vater such as pyloric stenosis.

Dr. E. S. Stuckey (Sydney) read a paper entitled "Fluid and Electrolyte Problems in Infants and Children: Intestinal Obstruction" (see page 205).

Professor Lorimer Dods said that he felt very hesitant about accepting without certain qualifications Dr. Stuckey's confident reliance on urinary chloride estimations, even when such tests were applied to all available specimens of urine. He would be interested to hear Dr. Macdonald's comments on the problem.

Dr. W. B. Macdonald (Melbourne) said that he would like to emphasize the fallibility of relying too much on estimations of urine chloride concentration as an index of plasma chloride levels. After all, chloride was a passive anion and the value of chloride estimations was to give a lead to the more important sodium concentrations, on which extracellular and plasma osmolarity and volume depended. If chloride and sodium were lost from the extracellular fluid *pari passu*, urine chloride concentration was a reflection of sodium loss, and also in states of alkalosis exemplified by pyloric stenosis, the presence of chloride in the urine after infusion indicated usually that the plasma concentration had been restored. However, in acidotic states, especially when sodium loss was severe, chloride would continue to be excreted in the urine despite a very low plasma concentration and severe hypoosmolarity, and would completely mislead the clinician who placed reliance on the urine chloride estimations. As diarrhœa was a disorder which resulted in this situation, an understanding of this was important to pædiatricians. This error of interpretation was quite distinct from added errors of biochemical technique.

Whilst they were on the subject of urine testing, it was well to realize that sometimes severely alkalotic infants with pyloric stenosis excreted urine which was acid in reaction.

Dr. D. G. Hamilton (Sydney) asked whether, in view of the excessive loss in chlorides and the development of alkalosis and pyloric stenosis and the value of stronger saline solution in its treatment, it would be good therapy to use such solutions initially in the treatment of any child in whom vomiting was a prominent part of the clinical picture.

Dr. H. Williams, in reply to Dr. Hamilton's question as to why a baby with pyloric stenosis developed alkalosis and yet a baby with vomiting and no obstruction did not, said that the cause of alkalosis was the loss of considerable quantities of chloride in excess of sodium. In pyloric stenosis there was often hyperchlorhydria with gastric retention, and these features rarely, if ever, presented with other causes of vomiting. Therefore in pyloric stenosis the infant often lost considerable quantities of chloride owing to vomiting of a retained secretion containing considerable quantities of hydrochloric acid. It was this great loss of chloride in excess of sodium loss that was responsible for alkalosis developing.

Dr. S. E. J. Robertson (Sydney) said that he had recently seen a child of four months with whooping-cough who had developed paralytic ileus and was treated with gastric suction. An attempt was made to replace the sodium fluid lost without avail and the child died quickly. Should he have started with potassium, and if so, how much should he have used?

Dr. E. S. Stuckey replied that in his opinion no potassium should have been used until adequate fluids had been given. Possibly the child did not have enough fluids to reestablish kidney function.

Dr. D. Galbraith (Melbourne) said that he wondered if this was a completely adequate explanation. Professor Stanley Graham, of Glasgow, had described an infant who, by chance, had been found to have pyloric stenosis before there had been any real vomiting. Yet this infant had biochemical evidence of alkalosis. Professor Graham has postulated a possible local chloride-controlling mechanism in the neighbourhood of the pylorus.

Dr. F. Arden (Brisbane) also asked whether it was possible to supply by the parenteral route alone a child's total caloric needs—using, for example, serum 20 mils per kilogram, "Cashydrol" 40 mils per kilogram and the remaining Calories as glucose. If this was given as 5% solution a considerable excess of water would be required. Did this matter? Dodd and Rapoport claimed that it was not harmful if the salt intake was controlled.

Dr. W. B. Macdonald (Melbourne), in reply to Dr. Arden, said that the provision of adequate Calories by parenteral means was difficult. In an endeavour to increase the glucose intake one often provided glucose more rapidly than the infant could metabolize it, with the result that it was excreted in the urine and took water with it by osmotic diuresis, thus counteracting the hydration process. As the maximum sparing effects of glucose were provided by less than a 5% solution, it was advisable not to increase the concentration above this level, although usually not much more than 200 Calories per day were provided in most cases. The intravenous use of amino acids was uneconomical, as by a combination of renal excretion as amino acids and gluconeogenesis approximately 85% was lost, only 15% being left to provide useful amino acids. Work on this had recently been done in America. In most cases it was better to neglect nutrition for hydration in the expectation that intravenous therapy would not be of long duration and that oral feeding would soon be resumed without the development of serious malnutrition. However, the problem was a difficult one.

Dr. M. L. Powell (Melbourne) asked what concentration of glucose in solution was advisable.

Dr. W. B. Macdonald, in reply to Dr. Powell, said that the concentration depended on many factors—the size of the infant, rate of metabolism of glucose, and rate of administration, to mention a few. In the case of one infant weighing approximately 16 pounds at the Children's Hospital, Melbourne, in the previous year, the rate of infusion had to be at least four ounces per hour, but even when the glucose concentration in the infusion fluid was reduced to 2·5%. glycosuria had been present.

Dr. H. Williams (Melbourne) said that it was important to consider the qualitative estimation of the gastric content, especially in various surgical conditions.

Dr. E. S. Stuckey said that fluid aspirated from the stomach in these conditions was approximately equivalent to N/2 saline. In newborn children it was probably less or likely to be at N/4 saline. He considered it safe to replace the estimated weight loss with fluid corresponding to half-strength physiological saline.

Dr. D. G. McKay (Adelaide) asked what place there was in the treatment of these conditions for the administration of blood serum.

Dr. W. B. Macdonald replied that serum was probably more economical than amino-acid preparations, because it was not lost in the urine and also contained electrolytes at a normal plasma concentration and pattern. However, the risk of homologous serum jaundice, estimated in one centre as being 2%, did not seem to justify the use of serum for nutritional purposes, as the risk of liver failure from protein deficiency was less. In the absence of this hazard, it would appear to be preferable to amino-acid preparations.

Professor Lorimer Dods said that if he was a good chairman he would proceed to sum up the morning's discussion; unfortunately he did not feel capable of even attempting this, so he would conclude with a few rather vague generalizations.

Firstly, at the risk of offering a glimpse of the obvious, he would emphasize the need for a reasonable understanding of the basic physiological principles involved in the maintenance of homœostasis. With this simple understanding and little or no knowledge of its more intricate aspects, they should be able to deal at least reasonably well with most of the problems as they met them in practice. Without such an understanding, any rational approach was obviously impossible and they found themselves back in that frightening world of ex-cathedra statements and rule-of-thumb methods. He felt that it was most important that both the student and the general practitioner should be carefully protected from all unnecessary and confusing teaching in this particular field and given instead some simple fundamental principles which they could understand and apply in their own practice.

The family practitioner and also the pædiatric consultant who was called to a consultation in the country or in a small hospital should be able to make quick and reasonable decisions about fluid and electrolyte therapy without the aid of detailed printed instructions or normograms. As many of those present knew from practical experience, the pædiatric consultant, when he returned from a consultation of this nature, had to be able to answer, with a minimum of hesitation, telephoned questions about the appropriate amounts and types of fluid to be given.

Just exactly thirty years previously Dr. Margaret Harper had visited the Newcastle Hospital and talked to Professor Dods and others, as members of the resident medical staff, about the value of subcutaneous and intraperitoneal saline therapy in the treatment of acute gastro-enteritis during infancy and had also mentioned the intravenous administration of saline to those infants. Dr. Harper's visit had made a tremendous impression on Professor Dods, as this was the first time he had heard anyone discuss these vital problems in any detail, for this was 1923 and they were just emerging from that dark dehydration era so aptly described by Professor Dunlop while he was lecturing in Australia: ". . . and when his thirst was unbearable they mocked him with 6 ounces of saline given per rectum." Although they had obviously come a long way during the past thirty years, it was most important that they should be very humble about their present sketchy knowledge of fluid and electrolyte problems and aware of the fact that in a few years' time they might be smiling at much of the electrolyte dogma of today.

Speaking in Australia in 1948, Sir James Spence had suggested that their knowledge of the chemical anatomy of the body at that time was probably comparable to the knowledge of physical anatomy in the time of Vesalius. Professor Dods thought that most of those present would agree with him that despite all the advances of the last five years the same statement could be made today with little fear of contradiction and this significant admission should help to remind them of their relative ignorance and of the need for humility in all these things.

Idiopathic Myocarditis in Infancy and Childhood.

Dr. Howard Williams (Melbourne) read a paper on idiopathic myocarditis in infancy and childhood.[1] After short reference to the historical aspect since the condition was first described in 1899 by Fiedler in an adult patient, he said that over the previous three years, August, 1949, to December, 1952, 14 patients with idiopathic myocarditis had been studied, twelve of them at the Children's Hospital, Melbourne, and two in the Hamilton Base Hospital, Victoria. All patients had been carefully observed clinically, and electrocardiographic and radiological studies of the heart had been made in most cases. In those patients who died routine macroscopic and microscopic examination had been made of all organs, and histological study of the heart included sections from both ventricles, both auricles, the interventricular septum and the valves.

Classification served as a basis for presentation of the patients rather than as a means of separating them into definite groups with distinctive clinical and pathological phenomena. Clinically every variant had been seen from severe acute cardiac failure, which was frequently rapidly fatal, to less severe more insidious subacute failure.

Dr. Williams discussed the patients in two groups: (i) The acute type. Nine cases had been characterized clinically by the sudden onset of severe cardiac failure. The pathological changes in the seven patients who died had consisted of widespread myocardial damage and interstitial cellular inflammation. The two surviving patients had recovered completely. (ii) The subacute and chronic type. Five cases had been characterized clinically by a rather longer history and more gradual development of cardiac failure. The pathological changes in the one patient who died were essentially the same as those in the acute group. The four surviving patients all had permanent cardiac damage.

Dr. Williams discussed the acute type in some detail. He said that there had been nine patients in this group, the ages ranging from twelve days to five years. The initial manifestations of the malady had commonly been mild, the mother attributing them to a minor ailment. Some patients had slight fever and were irritable and difficult to manage, others were vaguely not well, while some refused their food, vomited or had loose stools. One had a sore throat, another a "cold". None of the patients had been in contact with infectious diseases and the parents and other members of the family were well at the time when the child became ill. These symptoms had been present for a few days or a week before the onset of cardiac failure, when the child rapidly became seriously ill. Circulatory and respiratory embarrassment were the oustanding clinical features. Pallor was a striking feature and varying degrees of cyanosis had been noted in all cases. Some patients were restless and had a rather apprehensive look, while others were apathetic. The skin was cold, frequently mottled, often clammy and occasionally wet, and the temperature was subnormal, normal or slightly elevated. The respirations were rapid, often as high as 90 to 100 per minute, usually deep with a short expiratory grunt or slight wheeze. The chest was frequently barrel-shaped from pulmonary emphysema. The percussion note over the entire chest was hyperresonant and at times the upper margin of liver dulness was displaced downwards owing to the low position of the diaphragm. The breath sounds were harsh and in some patients crepitations were heard at the lung bases. The pulse rate was rapid, usually between 150 and 170 per minute, the pulse weak and thready, and the blood pressure low. Cardiac enlargement was always present, but often could not be detected clinically as the rapid respiratory rate and restlessness of the infant made palpation of the apex beat impossible, and the emphysematous chest rendered percussion of the heart unreliable. Sometimes cardiac enlargement was suggested by palpable pulsation immediately below the xiphisternum in the epigastrium. The heart sounds were usually of poor quality, the second sound at the apex often being louder than the first, and in some gallop rhythm further indicated cardiac embarrassment. Cervical venous engorgement could not be determined with any degree of certainty because of the short stumpy neck and restlessness of most infants. The liver edge was invariably three or four fingers' breadth below the costal margin, but could be overlooked unless palpation was commenced well down in the lower half of

[1] This paper will be published in full in *Archives of Disease in Childhood.*

the abdomen below the liver edge. The spleen had been palpable in a few patients. Subcutaneous œdema was present in only two patients and in three cases the urine contained a trace of albumin but no red blood cells or casts. The overall clinical picture was that of an anoxic infant with severe circulatory failure and considerable respiratory distress. The clinical features of gross liver enlargement and minimal pulmonary congestion suggested that the cardiac failure predominantly affected the right ventricle.

Radiological examination of the chest had shown gross uniform cardiac enlargement. In contrast to the large heart the lung fields were usually not congested, but were often hazy; this appearance suggested some œdema. In several cases small bilateral pleural effusions were noted, and one patient had an area of radioopacity at one lung base suggesting either collapse or consolidation. The electrocardiogram showed serious impairment of the myocardial function, the significant abnormal features being low voltage *QRS* complexes, especially in lead I, and low, isoelectric or inverted *T* waves in all three standard leads.

Necropsy showed that all hearts were dilated and the walls were usually thicker than normal. The only other macroscopic abnormalities were flabbiness of the heart muscle and irregular reddish or pale streaking in the muscle wall. With one exception the pericardium, endocardium and valves were all normal. Histological examination revealed widespread but often patchy damage of the myocardium, and an associated inflammatory reaction with neutrophile and eosinophile polymorphonuclear leucocytes, lymphocytes and macrophages.

In discussing subacute and chronic myocarditis, Dr. Williams said that, in contrast to the group of nine patients with an acute illness and rapid onset of severe heart failure, five patients had an illness of more gradual and insidious onset before cardiac failure developed. Four of these five patients had recovered, but all had residual myocardial damage. The prodromal symptoms were those of general malaise or vague ill health for a month or more before symptoms directly referable to myocardial failure developed. The outstanding and constant symptoms of failure were vomiting, rapid grunting breathing, pallor, cyanosis and later œdema. The physical signs present in all were cardiac enlargement, tachycardia, gallop rhythm, gross liver enlargement, and by contrast minimal signs of pulmonary congestion. Radiologically there were gross but symmetrical cardiac enlargement, hazy and slightly congested lung fields and often small bilateral pleural effusions. The electrocardiographic findings were the same as in the acute type. The *P* waves and *P-R* interval were normal, the amplitude of the *QRS* complex was low in all leads, but especially in lead I, and the *T* waves were flat, isoelectric or inverted. The course of the disease was prolonged. All patients responded to digitalis therapy, but had relapsed at varying intervals following its cessation. All had permanent cardiac damage, and in two there was evidence of a mitral valvular lesion. The pathological lesions in the patient who died were essentially the same as those in the acute group. While it was not possible to prove that the pathological lesion in the four survivors was the same, the clinical, radiological and electrocardiographic features were identical.

Regarding diagnosis, there were two main reasons why myocarditis was rarely diagnosed clinically. First, it was generally believed to be a very uncommon disorder and therefore was rarely considered. Second, the pattern of acute cardiac failure in a young infant could be a confusing one unless encountered previously. Distressed rapid breathing, cyanosis, cold extremities, poor capillary circulation and tachycardia occurred in both cardiac failure and severe acute respiratory tract infections, especially pneumonia. Failure to demonstrate cardiac enlargement was the most important reason for mistaking acute heart failure for pneumonia. It was often impossible in a restless infant with a respiratory rate of 50 to 100 per minute to determine the size of the heart clinically, and in such a patient radiological examination was necessary. Other features such as low or normal temperature with tachycardia, poor quality heart sounds and gallop rhythm, and especially a liver edge palpable three to four fingers' breadth below the costal margin, pointed to cardiac failure and not pneumonia. It was to be remembered that acute severe bronchiolitis with obstructive emphysema might cause rapid breathing, cyanosis and downward displacement of the liver. The displaced liver in such a patient might easily be mistaken for an enlarged liver.

Paroxysmal tachycardia often gave rise to heart failure if the heart rate was very rapid, and the attack was of some days' or weeks' duration. The chief features differentiating the two conditions were the heart rate and alteration in the ventricular complexes and *T* waves. In myocarditis the rate was rarely above 160 to 170 per minute, while in paroxysmal tachycardia with cardiac failure in infants it was rarely below 200 per minute. An electrocardiogram would usually establish the diagnosis, but it had to be remembered that Lind and Hultquist had shown that myocarditis might precipitate an attack of paroxysmal tachycardia.

Respiratory distress, cyanosis, and cardiac enlargement in a very young infant might be attributed to heart failure due to a congenital malformation of the heart. The heart failure might have been precipitated by a respiratory tract infection. Differentiation from heart failure due to myocarditis might be difficult, but the following points helped to establish the diagnosis. The infant with malformation of the heart usually did not thrive, often had mild attacks of cyanosis, and might be found to have an enlarged heart and murmur some time before cardiac failure developed. In contrast to myocarditis pulmonary engorgement was usually gross, the liver was not as large, the heart sounds were usually louder and often a murmur was present. Gallop rhythm was rarely heard and the electrocardiographic changes rarely showed evidence of gross myocardial damage.

It might be considered that the four patients with subacute myocarditis who recovered and especially the two who developed an apical systolic bruit had not had idiopathic but rheumatic myocarditis. Such a diagnosis was highly improbable on the grounds of the very young age of the patients, the complete lack of other clinical manifestations of rheumatic infection and the absence of both pericarditis and endocarditis during the active phases of the disease when congestive failure was present. In addition the electrocardiographic changes with grossly abnormal *T* waves and low amplitude ventricular complexes, and the repeatedly low antistreptolysin titres recorded in the serum were features which were rarely encountered in rheumatic carditis.

Turning to treatment, Dr. Williams said that infants with cardiac failure from myocarditis were dangerously ill and gentle nursing and minimal interference were essential. No attempt should be made to feed them until congestive failure was relieved and they should be left undisturbed in a warm cot or bassinette in an atmosphere of oxygen. Even minimal handling might lessen the chance of recovery; therefore cooperation and understanding by the nurse in charge of the infant were essential.

The condition should be rapidly brought under control with digitalis. Two patients in the acute group and all in the subacute group had responded promptly to the drug so that within twenty-four hours there was very appreciable improvement, and by seventy-two hours most of the signs of failure had disappeared. The dose used had been one-quarter to one-fifth of a grain of digitalis folium per pound of body weight, or 0·04 milligramme of digoxin per pound of body weight over a period of twenty-four hours. One-third of the total dose might be given immediately and the remainder in three evenly divided doses, at six-hourly intervals. While some patients had been treated satisfactorily with digitalis by mouth during the first twenty-four hours, it was wiser to use digoxin intramuscularly to obtain a more rapid effect. Oral therapy seemed quite satisfactory after the first twenty-four hours. Digitalis should be continued with a maintenance dose of approximately one-fortieth of a grain of digitalis folium per pound of body weight until the heart had returned to a normal size or the cardiac lesion had healed. It was difficult to decide when healing had taken place, and as cardiac failure in all patients in the subacute group recurred after cessation of several weeks' treatment, it seemed advisable to continue digitalis for a minimum period of three to four months.

Antibiotic drugs had been administered to some patients. There was no evidence that penicillin either alone or in combination with streptomycin or aureomycin altered the course of the illness.

The prognosis for the infant or child with a short history and severe circulatory failure was poor. The majority had not responded to digitalis therapy and were dead in less than twenty-four hours from commencement of treatment. However, two seriously ill patients had recovered completely and it was possible that earlier diagnosis and treatment might aid in the recovery of more patients in this group. All patients with the subacute type of illness had made a satisfactory initial response to digitalis, four of them survived but had enlarged hearts and two had a mitral valve lesion. The development of a valve lesion was not surprising, as one patient who died had infiltration of the atrioventricular ring and the base of the mitral valve with inflammatory cells. The two patients with a valve lesion

had been observed for over two and three years respectively, were in good health, and the cardiac lesion seemed stationary. Observation over years would be necessary to learn what would happen to these patients. It was possible that young adults with cardiac enlargement of unknown ætiology and with varying degrees of impaired cardiac function had developed their disease in infancy or early childhood and that it was similar in type to the disease in these patients.

Dr. Williams drew the following conclusions.

1. Idiopathic myocarditis was a serious, frequently fatal, malady occurring usually in the first two years of life.

2. The pathological lesion consisted of widespread but usually patchy muscle damage with an associated inflammatory cell reaction.

3. The common clinical illness was a rapidly developing, usually fatal, cardiac failure which was manifested by severe respiratory embarrassment, cyanosis, tachycardia with low or normal temperature, an enlarged heart and liver, and electrocardiographic changes of myocardial damage.

4. The illness in some patients might be more insidious in onset and the heart failure not as severe. Recovery frequently occurred after a long illness, but there was permanent myocardial damage.

5. The acute form of the disorder was commonly mistaken for pneumonia as respiratory embarrassment and cyanosis were the outstanding clinical features, and cardiac enlargement was difficult and frequently impossible to detect clinically.

6. Treatment with digitalis and oxygen had been of very definite value in some cases.

7. The clinical and pathological evidence gave no clue as to the ætiology.

Dr. Kate Campbell (Melbourne) reported a small epidemic of infective myocarditis in a neonatal nursery in a community hospital. There were six cases, and one of the patients died. In two cases the myocarditis was complicated by meningitis and in others by enteritis and nephritis. Digitalis did not appear to have any effect on the cardiac condition, nor was the course of the disease influenced by drugs.

Heavy colds or "influenza" in the mother at the birth of the baby might cause the infant to be born with myocarditis. Dr. Campbell had seen one such case in which an infant had clinical signs of myocarditis and died when thirty-six hours old. At autopsy acute myocarditis was found in microscopic section. A similar case, also proved by post-mortem examination, occurred in a patient under the care of a colleague. A third type of infection was that of an infant whose mother had a heavy cold with a temperature of 102° F. about the third post-partum day. The infant developed signs of an overwhelming acute myocarditis and post-mortem histological examination showed this to be present.

Professor Lorimer Dods asked Dr. Williams if he would explain why these babies died of congestive heart failure. Dr. Williams said that Dr. Powell would answer that question.

Dr. M. L. Powell (Melbourne) said that this was slightly speculative. The hearts that Dr. Williams had shown were so enormous that it seemed reasonable to suggest that the inferior *vena cava* was so distorted by the grossly dilated heart that pooling of the blood in the liver occurred rather than in the lung fields. Also it was probable that gross functional tricuspid incompetence occurred owing to the gross atony of the heart. Dr. Powell said that he would like to ask Dr. Williams one question. In the patients noted to have a mitral murmur, was the heart still big at that time and did he think that that murmur might have been produced by simple cardiac dilatation rather than actual valvulitis?

Dr. H. Williams stated that the two children in whom a murmur developed had had the cardiac lesion for some weeks or months and that the heart was enlarged at the time the murmur developed. The clinical evidence of the type of murmur and its persistence, unchanged over several years, suggested that the cause was valvulitis and not dilatation of the atrio-ventricular ring. In one patient who died the pathologist had found evidence of valvulitis; the mitral ring and base of the valve cusps were infiltrated with inflammatory cells.

Dr. M. T. Cockburn (Adelaide) asked Dr. Williams whether, in the absence of congestion in the lung fields, splenomegaly was apparent in any of these cases on clinical examination. He knew of at least two infants in whom there was definite splenomegaly as well as hepatomegaly.

Dr. H. Williams stated that in three of the acute cases the tip of the spleen was palpable and in one patient in the subacute group.

Dr. W. B. Macdonald (Melbourne) asked whether the emphysema made the pulmonary congestion less prominent.

Dr. H. Williams replied that all the children had emphysema and that it was probably due to the long-continued rapid respirations of 60 to 80 per minute. The emphysema would mask any pulmonary congestion. He also thought that in this type of heart failure it was predominantly right-sided heart failure and that pulmonary engorgement was not a prominent feature in that type of failure.

The Minor Epilepsies in Childhood.

Dr. D. G. Hamilton (Sydney) read a paper entitled "The Minor Epilepsies in Childhood" (see page 207).

Dr. Felix Arden (Brisbane) remarked that it had been said to be necessary to watch the blood count and urine. He asked what particularly was to be looked for in the urine.

Dr. D. G. Hamilton said that albumin should be looked for. A few cases of nephritis had been reported which apparently resembled in every respect early type 2 nephritis. He thought that in all cases this nephritis had resolved fairly rapidly when the administration of the drug was stopped. As far as the blood changes were concerned, they were important but not common. He had seen three or four cases in which the granular cells had fallen to a level causing concern.

Dr. M. T. Cockburn (Adelaide) asked, in view of the importance Dr. Hamilton had placed on the electroencephalogram, what was the age group of the patients whom he had in mind. It was a question which exercised Dr. Cockburn; in Adelaide they had only recently had available an electroencephalograph and the person who did the work for them was unwilling to prepare a tracing from anyone under the age of seven to eight years because he felt he was not able to give a particularly reliable report.

Dr. D. G. Hamilton said that in young children the electroencephalogram was less stable than in those who were older; rhythms tended to be much slower and it was common to obtain a borderline result. The rhythm in an epileptic infant was less regular and slower than one tended to find in a normal young child and one did sometimes find paroxysmal activity, spikes and waves in the tracings of the former and this was abnormal. It did not mean that the child was epileptic, but it did indicate a brain abnormality. It was important to be experienced in electroencephalography and the results were less reliable in a young child.

Dr. M. L. Powell (Melbourne) asked about the presence of abdominal pain in such patients.

Dr. D. G. Hamilton mentioned one patient who had abdominal pain and colic and was very sleepy. This child had an abnormal electroencephalogram.

Dr. J. H. Colebatch (Melbourne) asked about the problem of prolonged hæmatological control of children on tridione. In the important initial period control was easy to ensure, but in both private and public hospital practice he periodically found a patient who had been carrying on therapy for months without any clinical or hæmatological supervision. He wondered how often, if ever, serious damage to hæmopoiesis occurred after the first two or three months.

Dr. D. G. Hamilton said that Lennox believed that in the first month the blood count should be done every fortnight, and in the first year, every month. Dr. Hamilton thought that it was worth while to make the patients aware of the possible symptoms. If these occurred, the patient should be brought for examination.

Idiopathic Hypoparathyroidism.

Dr. Felix Arden (Brisbane) read an article entitled "Idiopathic Hypoparathyroidism" (see page 217).

Dr. H. Williams (Melbourne) asked Dr. Arden whether any patients he had read about had had complications from prolonged hypercalcæmia.

Dr. Arden said that he had not read of any such case, though physicians had been very worried about this, as there was a tendency for the calcium content to fall to a low level of normal. It was necessary to have a high blood calcium content for a long period of time.

Dr. D. Galbraith (Melbourne) said that Albright had described this condition quite fully. One point that Dr. Galbraith wanted to mention was that in one of his cases he stressed the fact that infection had been the underlying

cause. One patient had pyelonephritis; this cleared up and the hypoparathyroidism cleared up also.

Dr. F. Arden said that in the histories of children treated it was shown that five of 52 had had some sort of virus disease.

Vitamin D Resistant Rickets.

Dr. Douglas Galbraith (Melbourne) read a paper entitled "Vitamin D Resistant Rickets" (see page 208).

Dr. M. L. Powell (Melbourne) said that his interest in refractory rickets arose from a remarkable family in which the grandmother, one of her sons, one of her daughters, and the four children of that daughter all manifested rickets. Dr. Powell had been in charge of the four children, and was interested from the point of view of dosage of vitamin *D* used in their therapy. The first two of these children reached a dosage of 50,000 units per day which seemed to control their rickets, though osteoclasis was required ultimately in both of them to correct the bowing. The third child whose skiagram showed evidence of rickets was put at nine months of age on the same dosage as the other two children—50,000 units per day. Within a month she manifested polyuria, an irritable skin, loss of weight, hypotonia, anorexia, and extreme irritability. She looked in fact extraordinarily like a patient with pink disease. Her serum calcium and phosphorus contents rose to approximately 16 and 18 milligrammes *per centum*, and it was quite apparent that she was suffering from hypervitaminosis *D*. The vitamin therapy was, of course, stopped, and finally a maintenance dose of 25,000 units per day was reached, and it was of interest to note that the rickets in this child had healed so well that the legs were perfect in shape and would certainly require no osteoclasis.

The fourth child, the baby, of some fourteen months now, was being slowly worked up in its dosage, to try to find an optimal figure. It was not without interest that this baby had reached 100,000 units daily without signs of toxicity.

Dr. Powell stressed the point that, in view of Dr. Galbraith's paper, this might be the wrong approach to this type of refractory rickets, and that it might be possible by others means, such as the addition of the citrate mixture, to control the rickets readily without such high dosage.

Imperforate Anus.[1]

Dr. F. D. Stephens read a paper entitled "Imperforate Anus". He explained that the term "imperforate anus" was frequently used loosely to cover all forms of rectal deformities, whether the terminal part of the bowel ended at a high or low level in the pelvis, and whether it opened to the urinary or genital tracts or to the skin by fistulous communications. The usual blindly ending rectal deformities had been found in his study to end at more specific levels, and the congenital rectal fistulæ were of constant types, arising primarily from the rectum at those specific sites.

The three special anatomical levels in the pelvis were: (i) At the pubo-coccygeal line. In the pelvis, bisected in the sagittal plane, this line joined the upper surface of the pubic symphysis with the sacro-coccygeal junction. This line passed through the upper margin of the verumontanum, the external os of the cervix, the peritoneal pouch in the male, and Houston's third fold of the rectum. (ii) The upper surface of the *corpus spongiosum* of the bulbous urethra. (iii) The caudal and posterior aspect of the *corpus spongiosum*.

In regard to X-ray levels, Dr. Stephens said that the clinical recognition of these levels could be determined in the totally imperforate condition on the lateral X-ray views (Wangensteen and Rice inversion method), care being taken in the radiological technique to centre the X-ray tube over the head of the femur, and at least one film being taken from a distance of six feet to prevent distortion of the ischial bones. The pubo-coccygeal line could be drawn in on the X-ray film as the ossific centres of the pubes threw a shadow over the site of the symphysis, and the fifth or sixth ossified sacral vertebra gave the level of the sacro-coccygeal junction (the coccyx ossified at two years). The upper surface of the bulb was at the level of the inferior margin of the ossified ischial bone, and the lower surface was approximately 1·0 centimetre caudal to this point.

Discussing embryological levels, Dr. Stephens said that the usual conception of the embryology would lead one to expect the terminal blind end of the rectum to end at any haphazard level in the pelvis between the upper surface of the endodermal cloaca and the skin. One would expect an ever-increasing number of cases of recto-urethral fistulæ in the lower half of the posterior part of the urethra owing to the persistence of the so-called cloacal duct which was the last part of the cloaca to be divided off by the down-growing uro-rectal septum.

The more specific levels of the common terminations of the rectum, together with the finding that there was a great rarity of recto-urethral fistula between the level of the verumontanum and the upper surface of the bulb, demanded a slight modification in the embryological concept. It was presumed, therefore, that, instead of the subdivision of the cloaca occurring by a continuous downgrowth of a uro-rectal septum, it was divided by two processes, one which separated the hindgut from the allantois above the level of the Müller's tubercle (verumontanum), and the other which divided the cloaca below the tubercle by lateral ingrowths of mesenchyme, meeting in the mid-line first at the tubercle and extending caudally to the cloacal membrane.

Failure of development of the second process in its entirety would account for the more common rectal deformities at the level of the pubo-coccygeal line and Müller's tubercle. Completion of this process, but failure of development of the anal pit, explained the appearance of the terminal end of the rectum at the level of the junction of the membranous urethra with the bulbous urethra.

The third and lowest level of termination of the rectum was explained by the formation of the perineum between the urogenital and anal pits. These two pits at first lay adjacent to one another. Owing to the development of the perineum, the anal membrane in contact with the rectum above, and anus below, migrated posteriorly and caudally to its normal site. Persistence of the anal membrane caused the blind terminal part of the rectum to lie at the usual site of the anal valves.

Lack of perineal development, or failure of migration of the anus, or overgrowth and posterior fusion of the inner genital folds might cause the anus or anal membrane to be covered in partly or completely on a more superficial plane or might project the anal lumen as a fine fistula anteriorly along the median raphé of the perineum even as far as the frenulum, or on a deeper plane into the bulbous urethra. The level of the rectum varied in this group between the upper and the lower levels of the bulbous urethra.

In regard to terminology, the names suggested for these deformities were: (*a*) Imperforate rectum with or without "recto-urethral" or recto-vaginal fistulæ for lowest level or higher level lesions. (*b*) Imperforate anus for blind second level lesions. (*c*) Imperforate anal membrane and anal stenosis for third level conditions, and ectopic anus for the anus otherwise normal except for its anterior position. (*d*) Covered anus for terminal gut sites varying from the upper to the lower levels of the bulb, but combined with a fine fistulous track to the bulbous urethra or to the mid-line of the perineum along the raphé in the male or the fourchette in the female.

Examination of specimens of these deformities revealed that the *levator ani* muscle, though abnormally arranged to conform to the visceral anomaly, was the best developed muscle of the sphincters in imperforate rectum deformities. In the imperforate anal membrane type all sphincters were present, though the external lacked its characteristic subdivisions, but the deep circular band could be identified. In the covered anus group the *levator ani* and internal sphincter were well formed. The external sphincter, in its deep circular part only, was present. No specimen of the imperforate anus at the level of the upper surface of the bulb was available for detailed examination, but observation after surgical operation suggested that the *levator ani* sling was adequately developed.

Finally Dr. Stephens said that treatment with a view to establishing an adequate anal orifice with sphincter control depended on the anatomy of the sphincter muscles in each deformity.

The sacro-coccygeal approach through the supralevator space was recommended for the imperforate rectum series. The new canal was directed to the perineum within the sling of the levator muscle.

The second level group of deformities could be treated by perineal approach, provided the incision and new anus were made close to the bulb. By making the incision anterior to the normal site of the anus, it was hoped to avoid cutting the lower sling-like fibres of pubo-rectalis muscle which curved in, hammock-fashion, under the blind end of the gut.

The imperforate anal membrane group required incision of the membrane and dilatation. When the membrane was

[1] This paper will be published in full in *The Australian and New Zealand Journal of Surgery*.

thickened by addition of tissue derived from the genital folds in the form of a thick central raphé or tag, additional trimming might be required.

The covered anus group demanded the diminution of the fistula and the formation of the anus. Denis Browne had shown this could be brought about in the cutaneous fistula group by simply laying open the fistula. One blade of the scissors was inserted in the fistula and one on the skin of the perineum. By cutting the tissues along the caudal wall of the fistula posteriorly the track was opened to sufficient dimensions to permit the introduction of a size 18 urethral sound. The laid open tissues were separated by packing each day until healing had occurred and the anus was dilated gradually with larger sounds until the fifth finger could be introduced. At this stage, daily dilatations were done by the mother. The anus was somewhat untidy, but very efficient, and scarring was minimal.

The ano-bulbar fistula was commonly complicated by an atypical hypospadias deformity. The perineal or scrotal orifice of the urethra and fistula was also cut back in the above fashion, both the lower end of the posterior part of the urethra and the anus being exposed, as two orifices situated one behind the other. The mucosal bridge on the upper surface of the fistulous track was divided and the incision was laid bare by packing. The anus was dilated gradually. The urethra was reconstituted by a second operation at a later date by means of the Denis Browne technique.

Dr. F. Arden (Brisbane) asked whether it would not be safer to perform simply a pelvic colostomy as an immediate operation when the rectum was found to terminate at the pubo-coccygeal line.

Dr. F. D. Stephens replied that a colostomy was certainly the safer procedure in difficult cases, but that transverse colostomy was the better operation as it did not tie the pelvic colon down and so rendered subsequent operations easier.

Dr. Eric Goulston (Sydney) asked what was the optimum age for operation in the high level cases.

Dr. F. D. Stephens said that eighteen months was about the minimum.

Dr. J. Steigrad (Sydney) said that he had used the sacro-coccygeal approach several times; it was easier to work through the perineal approach. The perineal approach for high level imperforate anus was unsatisfactory. Four or five cases in which he had used the perineal approach for high level obstructions had shown how unsatisfactory it could be. He mentioned the case of a covered anus in a girl in which a so-called fistula opened in the posterior part of the vulva. He was interested to know how the condition healed. In two cases, one of low and one of high vaginal opening, he had moved the vagina back through the perineal incision; the passage now was very satisfactory. Both patients were able to continue school and did not soil clothes. He wondered how the condition appeared in the covered anus in the female after an ordinary cut-back.

Dr. F. D. Stephens said that the healed anus was somewhat untidy with tags of excess skin. On parting of the buttocks, the rectal mucosa was easily seen, indicating absence of the lower superficial sphincter muscles, but the children were very continent.

Round Table Discussion: Child Health Facilities in Australia in Relation to the Total Needs of Children.

A round table discussion was held on the child health facilities in Australia in relation to the total needs of children. Dr. F. W. Clements occupied the chair.

Introductory Paper.

Dr. F. W. Clements (Sydney) read an introductory paper. He said that the session was the outcome of the discussions at the previous annual meeting which culminated in the surveys that had been made of the pædiatric services in each State, copies of which had been distributed to members. He thought that they should not devote over much time to talking about the surveys, but should concentrate on the problems demonstrated by the surveys.

Dr. Clements discussed first the total needs of children. He said that over the previous fifteen to twenty years considerable changes had taken place in the concepts of what were the total needs of children for healthful living. These were clearly shown in the change in emphasis at kindergartens. Dr. Clements had first begun examining children at kindergartens about twenty years before. Then a typical request from the director would be for him to determine if there were any physical features which might account for the child's behaviour; this might be refusal of food, lack of interest in the centre's activities or general anti-social behaviour. Each of these had often been accompanied by failure to grow at a normal rate. He had generally managed to find something to blame, enlarged tonsils or some anæmia (verified by hæmatological examination) or clinical malnutrition, which he related to lack of some nutrient. In other words the emphasis had been on the somatic aspects of the child's life.

Today the attitude in progressive kindergartens was the reverse. When the medical unit found that a child was not gaining in weight at a satisfactory rate or was showing signs of poor nutrition (this word being used in its widest sense), after having verified the absence of any gross signs of disease, they turned to the kindergarten staff and social worker and asked what there was in the life of the child which was preventing it from living a healthful life.

This approach had been summed up in the preamble to the constitution of the World Health Organization: "Health is a state of complete mental, physical and social well being and not merely the absence of disease or infirmity."

It would be over-simplifying the issues to say that, for the child, mental and social well-being meant security; but this idea did help to direct attention to the importance of parents in the total picture. It was now recognized that in childhood, a sense of security, imparting, as it did, the ability to effect adjustment to changing conditions, was dependent upon good mother-child relationships. These in turn required emotionally stable and secure parents who combined a natural love for their children with a working knowledge of what was necessary for the development of mental, physical and social well-being. Dr. Clements said that he would refer to this later.

In regard to the structure of society, Dr. Clements pointed out that the child as seen in the playground, school, hospital, and out-patient department was the product of the society in which he lived. The Australian society was now predominantly urban, having crossed over from a predominantly rural population in the 1920's; since then the urban portion had been increasing rapidly. The characteristics of the two types were different.

The present was the era of small families. Parents tended to have an increased interest in their children; more people hoped and expected their children to grow and to develop into high grade citizens. A larger number of people knew what constituted good physical growth, and more and more were seeking information about what was necessary for the satisfactory development of their children and their adjustment into the home and the community.

Dr. Clements referred to the considerable changes in the patterns of health and disease which had occurred in Australian children over the last twenty-five to thirty years. Forty years previously the infectious diseases had been responsible for 70% of the deaths; today, with much lower death rates, they were responsible for less than 30%.

Consistently with the fall in deaths from infectious diseases there had been a drop in the incidence and severity of these conditions. It was worth noting, in passing, that with the fall in deaths due to bacteriological diseases other conditions had leapt into prominence as killers, for example, accidents, congenital malformations, and the more or less hopeless diseases like the leuchæmias and cancer.

Parallel with these changes, medical practitioners were also conscious of the developments in their own concepts of disease. They had seen the integration of the purely mechanistic concept of illness, which thought of illness as a breakdown of some part of the human machine, into the ever-expanding biological concept. Since the latter took into account man's reaction to his environment it had been widened with each passing decade as progressive additions had been made to the primitive climatic factors, the bacteriological and then the psychological, including in more recent times the effects of "social" environment.

Unfortunately, the integration of the mechanistic and biological concepts of illness had not yet found full expression in preventive medicine, medical care or medical education. If they had accepted this idea they would more frequently adopt Halliday's suggestion that instead of asking "What is the cause of this illness" they would ask: (i) "What kind of person is this?" (ii) "Why did he become ill when he did?" (iii) "Why did he become ill in the manner he did?"

The changing patterns of illness were going to require doctors to give more thought to some of the biological factors in ætiology. Many doctors reported a marked increase in the numbers of mothers who brought their children for consultation because of what they loosely

termed behaviour problems. A lively discussion could no doubt develop around the question of whether there had been an increase in the incidence of behaviour problems or whether parents were now more aware of them than before. Many competent students of medicine and sociology believed that there had been an actual increase, but there was also evidence to support the belief that parents were becoming increasingly anxious for their children to grow properly and for their satisfactory adjustment into the community. This would be in keeping with both the changing structure of society and the advances in parent education, to be referred to again in the discussion.

The changes in the pattern of disease had shown themselves in another way. When infectious diseases and what might be called general illnesses occupied a prominent place in the lives of the people and their doctors the emphasis had been on infectious disease hospitals, general children's hospitals and children's wards in general hospitals. In more recent years, interest had turned to the provision of services for handicapped children or for children with chronic or rare diseases. Some of these services were extremely expensive per child, for example, the full services for the crippled or spastic child.

The last point discussed by Dr. Clements was the philosophy behind the services that had been provided. He said that knowledge about health and healthful living did not just happen; although beliefs and practices were passed on from one generation to the next, each generation learned much from current teachings, sometimes, however, with some resistance.

A review of health education material used over the previous twenty-five to thirty years showed that in general each stage corresponded to the scientific activity and thought of the period. In the 1920's and early 1930's emphasis had been on the bacteriological diseases, on personal and community hygiene. In the late 1930's and 1940's nutrition had dominated the scene.

In more recent times many parents had broadened their horizons and were asking for help and guidance in assisting their children to live fuller and more useful lives.

Services, including pædiatric services, were in general the outcome of demands by the community or a section of it. Up to the present, with few exceptions, the services provided had been materialistic hospitals, playgrounds, kindergartens, medical care and the auxiliary medical services. Although primarily materialistic, many services had made some contribution towards the emotional and social lives of children.

Dr. Clements asked whether in their preoccupation with the materialistic issues they had concerned themselves too much with the personal welfare of the human being in their man-made society, and he thought that they had not paid enough attention to the important role played by parents in the lives of their children. In their review of the facilities available for the total needs of children that day they would do well to inquire whether this had affected the quality of the services rendered. If this had been so, then they should want to know in what ways they could make more effective use of parents, and conversely how they could give more effective service to parents, as parents.

Perhaps it was that the medical profession was not adequately equipped to meet the parents in this new role, and that because of their own deficiencies they preferred to have the parents out of any picture.

Provision for the Total Needs of Children at Home and in the Community.

Dr. David Jackson (Brisbane) confessed that it was only when Dr. Clements suggested that he should deal with the provision for total needs of children at home and in the community that he realized that there was a tremendous hiatus in the survey of child health facilities on which he had been working intermittently for more than two years. He had completely disregarded the facilities existing (or not existing) where the child spent most of his time and where his mental and physical future were moulded, namely, in his usual environment—home, school and playing fields.

Dr. Jackson could not pretend to speak as an authority on the subject, and he rejected the idea of making a belated effort to collect information about pre-schools, kindergartens, school medical services and so on. As Dr. Clements had pointed out, that was only part of the aspect he was trying to present; they were the services provided by the community, whereas Dr. Jackson felt that it was part of his task to give more emphasis to the services that were or could or should be provided by parents and families.

This had of necessity to be rather vague and speculative, and he thought that what was necessary here and now was to try to decide how to put these two components of the needs of the well child at home and in the community in their proper place beside the other information.

Dr. Jackson did not think anyone present would disagree with the importance of the aspect of child health under discussion. But why was it necessary for them to know about its present position and what should be done by them about it? What was their responsibility and what were their capabilities in the matter? What did they need to know?

In this survey as a whole they were considering needs and trying to find out how facilities fell short of filling needs. Possibly in the fields so far explored by the survey they all had a fair idea of what they would like in the particular field that interested them. Could they then start thinking (and Dr. Jackson for one had not got very far) about what they would like for their patients or for the children of Australia in their own natural environment?

Probably most of those present had a rather vaguely outlined idea of conditions as a whole in the areas covered by their own practices. Perhaps they could select one or two family groups in which conditions seemed nearly ideal and some at the other end of the scale with a vague mass of ideas about the families in between, and they could probably put together collectively a pretty formidable list of evils and ways in which things were falling short—living conditions that were bad for a variety of reasons, the tension due to neighbours, lodgers or "in-laws", restricted companionship, restricted play areas, mothers who had to work, the incubus of an abnormal child in the family, ignorance and the evil of "a little knowledge".

They probably all appreciated, even if they had not thought of it before, Dr. Clements's point about the loneliness of units in urban communities. But they had to remember that rural Australia was not entirely made up of communities such as he had mentioned, and there were families living in complete geographical isolation. In fact there must surely be many patterns, all over Australia and all demanding attention.

Now there might be (Dr. Jackson did not know) a lot of statistical information about this—densities of population, numbers of children per family, numbers of families per house and so on. But even if there was, he did not think it would help them very much. They wanted something more human and personal than that.

So, once again, could they begin to outline what they would like—of necessity in very general terms?

They might begin by saying that they would like at least a certain standard of housing, clothing and feeding, but it was the less tangible things that were more interesting. Dr. Jackson suggested (in the hope of stimulating discussion) that they would like to see conditions, mental and physical, that would give children a sense of security, protection, help and guidance—leading not to over-dependence but to the virtues of courage or steadfastness, integrity and loyalty—conditions which fostered the ability of the developing individual to mix easily with others of his own age, which allowed children to develop friendly relationships with adults and which avoided emotional disturbance or difficulty.

Implicit in that was an attitude on the part of parents of intelligent interest and enlightenment regarding the true welfare of their own family group, and that included a knowledge of what existed for their benefit and how to use it.

Finally, if they wanted (as Dr. Jackson thought they should) to put themselves in the picture, they could include the doctor who really knew and understood the family, who could see and observe the children frequently, who, because he knew and understood the normal, could detect trends towards the abnormal and knew what to do about them, especially a doctor who knew what to advise, and as part of that ability knew what facilities existed and how to use them.

Those present might or might not agree with what he had said, but underlying these suggestions, and Dr. Jackson thought underlying any suggestions, two needs became apparent—the need for the education of parents and the need for the education of the medical profession—which should itself include education in how to educate parents.

Turning to the "community" services aimed at reaching the normal child and fostering his mental and physical welfare, Dr. Jackson said that he could be very brief, for they were very few.

There were the maternal and child welfare services; there were some kindergartens and nursery schools in the larger centres, some playgrounds, and for the older groups the school health services, the schools themselves and some free-time organizations such as that of the Boy Scouts. These were the community services which they would have listed in their survey, and some of them had done so.

Was there anything else? Yes. There was the domiciliary preventive medical service provided by doctors, and Dr. Jackson was sure that they should include the part played by the churches in the lives of the people.

Why, where and how were these facilities or services failing? The very smallness of the effort in relation to the size of the problem was one obvious answer, but even the few that there were available were failing to reach the people they were designed to help, or to reach and help them in the right way.

In this connexion Dr. Jackson thought that they would do well to ask (and he did not know the answer) why the infant welfare services, though by no means perfect, were reaching a substantial proportion of the age group they were designed for (estimated at 60% in Queensland and nearly 100% in Tasmania), while others touched only the fringe (kindergarten and nursery schools in New South Wales catered for less than 10% of the children of that age).

Was there a natural instinct to resist (interference, or a pride that deterred people from accepting a service? Did the organization of services as such lead to the development of a sort of "crust" which stopped them from reaching the hearts of the people and stopped people from reaching them?

Was there a bit of humanity missing? Was it a link that the doctor could supply—the man who knew what existed, knew how it should be used, and knew how to put his "case" in touch with the appropriate facility?

What should be done?

Dr. Jackson said that he did not propose even to attempt an answer to that huge question, and he would tell those present why. It was because it was so fatally easy to plan, and he thought that they ought consciously to resist the awful temptation to become planners.

But perhaps he should now try to draw some of his discursive threads together. He had said that they should try to put this subject in its proper place beside their other information. To do that they needed a mosaic picture of family life and needed it all over Australia, and because they were doctors he suggested that they should try to obtain that through doctors—all over the country. To know what to do about it they had to have some sort of picture of what they wanted, and that picture would become more distinct as information about needs was obtained. To discover the needs and to help correct them required a new outlook to be instilled into the medical profession, which had to be a slow process. They could play their part in that through undergraduate education, but that was not enough. The newly qualified were absorbed and diluted by the profession at large, and so post-graduate education was necessary, too. Through the education of their own profession doctors could make a big contribution towards the great need of parent education.

As individual members of this association, their efforts would of necessity vary according to their individual outlook and interests, but the Association as a whole was more important. The Association could collect information and be a centre of information. It could keep in touch with developments and pass them on to members or hold them available for members, and in this respect he commended to those present the system of standing committees of the British Pædiatric Association.

The field was an enormous one, and there were all sorts of other people working in it—teachers, educationists, sociologists and so on. Pædiatricians could not, nor did they want to, do the jobs for others, but as an association they could see that the others did not do their jobs. As a pædiatric association they should be able to keep in touch with the others so that their help and information were through the Australian Pædiatric Association available to its members; and they should make sure that the others knew about the Association, knew that through the Association they might have the sympathy, interest and help of pædiatricians, and that in the Association they might have a source of expert advice.

Provision for Total Needs of the Sick and the Handicapped Child.

Dr. Eric B. Sims (Adelaide) discussed the total needs of sick, handicapped, or deprived children. He said that from the State surveys that had already been circulated among members it would appear that the number of hospital beds available in Australia for acutely sick children were more or less adequate for the communities they served, although there was still a need to "humanize" the care of children in hospitals in most centres. However, it was obvious that big gaps existed in the child health services of all States, notably in the field of mental health. There was also inadequate care of some handicapped children, especially those who lived in the country away from the facilities available in the cities for their particular problems.

Apart from these deficiencies it was probable that most Australian capital cities had just barely adequate provision at the moment for the physical needs of sick, mildly crippled, blind, deaf, "rheumatic" and tuberculous children.

In reference to the needs of sick children, Dr. Sims said that it was, of course, axiomatic that the aim of pædiatrics was to prevent illness and to avoid the admission of children to hospital whenever possible. Some sick children, however, would need hospital care, and his first task was to consider this aspect of child health in more detail.

The children's hospitals of Australia had probably all been conceived—as elsewhere in the world—between 1850 and 1880, and had been designed along the lines of the gloomy adult hospitals of that era. More recent additions to their fabrics had somewhat modified the large wards, the steps, corridors, and plumbing, but only in the last few years had the special needs of children been realized. What, then, did they want their children's hospitals to be like? A few basic principles might be stated: Ward units should be small, and include a number of smaller bedrooms, a playroom, a room for parental interviews, one for treatments and painful minor procedures, a doctor's room, a side laboratory, nurses' room *et cetera*. Children should be placed in wards or rooms according to age, temperament and the nature of their illness and rarely should be alone. Another basic principle that most of those present would accept was that the parents had to have reasonable access to their children in hospital, and that the mothers of infants under, say, three years, should be encouraged to participate as much as possible in their nursing. The logical end of this was the provision of units in which mothers and infants roomed together, and this, of course, had been found to be eminently workable elsewhere. The advantages of this arrangement, both physical and emotional, for all concerned needed no stressing.

Finally the hospital ought to be kept in touch with the community, and the episode of the child's stay in hospital should be fitted into the general stream of his continuing care by close collaboration between the hospital staff and his own doctor. As Professor MacIntosh had said: "The hospital must be a sphere of influence . . . reaching to the homes of the families it serves." No so-called health service could succeed if the hospital was isolated from the community, and if the institution was solely interested in the problems of the child when sick.

As to the effect of all this on the child, after infancy the average child, even in an old-fashioned children's hospital, was sufficiently resilient and stayed for a sufficiently short time to come to no permanent harm. The problem of the long-stay hospital was more dangerous. As Spence had said:

> Too often and too lightly is the decision made to confine children in long-stay hospitals. . . . A long-stay hospital can never be completely successful in providing the things of which the child is deprived, whatever the educational arrangements may be, or however many their books, toys and cinemas. These may mitigate the deprivation, but do not give the sense of personal attachment, the relationship, the companionship, which are necessary exercises for the mind of the growing child. . . . It would be better if the children lived in small groups under a house-mother . . . [whose duty it is] to live with her group of children and attempt to provide the things of which they have been deprived.

This raised the question of the care of children in institutions in general—whether for sickness, handicaps or parent-deprivation. It was basic that a child's home had to be very bad indeed to be worse than the average institution for children, and in many situations perhaps the aim should be, not to multiply the number of institutions, but to help and to guide the parents to care for their particular child at

home. However, presumably there would always be homes that could not, for medical or social reasons, cope with a handicapped child, and there would probably always be homes that would have to be accepted as too bad for children to live in—although Dr. Sims often wondered how many children, taken from such dirty environments by court order, were made happier and better citizens in the clean institutions that were the usual alternative.

The type of care that a child needed in a foster home, or a long-stay hospital, had been considered by the Curtis Committee in England, and more recently in a Home Office Memorandum. Both these documents should be studied.

Discussing handicapped children, Dr. Sims said that he had already indicated briefly gaps in the services for handicapped children. The various grades of mentally defective children, for instance, were inadequately catered for in most States, but this fact was realized and pressure groups, composed of parents and other interested parties, were slowly arousing the public conscience. No doubt, when a sufficient number of people demanded it, governments would act appropriately. However, it was highly desirable that the present system of individual "good works" by private enthusiasts should not go on undirected. Already in the community a large number of small charitable bodies could be seen nibbling at portions of the same problems, overlapping in some fields, leaving areas untouched by any of them in others. There was a real danger that sentimental considerations would lead to an undue emphasis on one particular aspect, to the neglect of other equally sad problems. For instance, although he was closely associated with the work of a spastic centre, Dr. Sims could not help feeling that cerebral palsy had become a fashionable disability and was almost unduly favoured in the attention it received at the moment. He found it very hard indeed to turn away children who were submitted for admission to the benefits of the centre, because they could not, by the most elastic interpretation of the term, be classified as suffering from cerebral palsy—especially when one knew that there were no other organizations available in the community to help the afflicted child and his parents.

This was a theme that could be developed considerably, but Dr. Sims said that he would leave it to the meeting to decide how to ensure that the efforts of the community on behalf of handicapped children could be made comprehensive and balanced. He realized that the initial endeavour must always be to provide help for the children with remediable disabilities. After all, there came a time when they had to ask themselves: "How much can a small nation afford to spend on its health services?" The natural corollary to this was the question: "And how much are we therefore justified in spending on the care of children who can never be productive citizens?" Such questions involved the community in moral issues that were not Dr. Sims's immediate concern, but at least it behoved them all to ensure that the large sums of money that were already being spent were being used wisely. Only a central planning and coordinating authority could do this—and only an association like the Australian Pædiatric Association could advise such a department.

Dr. Sims summed up his remarks with the following statements:

1. The physical needs of acutely ill children are probably provided for adequately by the children's hospitals in the capital cities. More regional pædiatric services might be developed in some country areas, but in most cases it will remain more economical to arrange transport to the cities for the specialized facilities of the base hospitals.

2. Humanization of the children's hospitals is necessary, and is under way.

3. Many long-stay hospitals do not conform to modern views on the total needs of the child. The number of long-stay beds available would probably be adequate if more thought was given to the matter before a child was committed to such places.

4. The needs of deaf, blind, rheumatic, diabetic and prematurely born children are generally realized, and are being (or will be) provided for in reasonable quantity and quality.

5. Residential accommodation in the city for country children who need various special facilities is inadequate.

6. Both a preventorium and a sanatorium for children have limited but definite value in the control of childhood tuberculosis.

7. Severely and incurably crippled children are not adequately provided for in most capitals.

8. Many mentally defective children (including severe spastics) are not helped in the existing set-up by appropriate institutions, occupation centres or advice. Even if money spent on such children is unproductive for the nation in the broadest sense, their parents would be better citizens—and perhaps have more children—if helped in their problems.

9. An advisory committee on child health is needed at the highest level to coordinate the provision of total child needs.

Education of Physician, Teacher, Parent and Other Workers in the Field.

Dr. Vernon L. Collins (Melbourne) dealt with the educational aspects. He said that in the round table session he had been allotted the responsibility of discussing the education of the physicians, teachers, parents, and other groups, in relation to the total needs of children. From the outset, however, he wished to state that he intended to discuss mainly the education of the medical undergraduate and graduate in this field, for it seemed obvious that if there were defects in medical training, medical practitioners should put their own house in order first.

In the opening paper, Dr. Clements had emphasized some of the changes in the structure of society and the changing patterns of disease which made it necessary to review pædiatric education achievements, and his question from a technical discussion group of the World Health Assembly was a pertinent one: "To what extent does the training of medical undergraduates and post-graduates prepare them to meet their social as well as their technical responsibilities?" Did the medical student receive adequate teaching upon the social and psychological factors in health and disease? Did he learn enough about man as a member of society, of problems of growth and development, and of preventive aspects of medicine?

At the meeting of the Australian Pædiatric Association two years before, there had been agreement, Dr. Collins thought, upon their two main aims in pædiatric teaching—firstly, the creation of a keen interest in pædiatrics, in its problems and its importance to the welfare of the community; and, secondly, the training of students in sound principles and practice of pædiatric medicine so as to fit them for family practice. Again, he thought that there was agreement upon the essentials which should be taught to the student to give a balanced training: (*a*) the factors necessary for health; (*b*) the common patterns of illness, their ætiology, diagnosis, prevention and treatment; (*c*) the initiation of the student into a way of intimately caring for his patients.

In the provision of total care for the child, the knowledge gained in training in these three principles was inseparable. The student had to know the normal and its variations, he had to be familiar with the management of the patient's illness, he had to know the psychological and social background, he had to understand the child's reaction to his illness and the mother's reaction to the child's illness. He had to be able to form a picture of the child in his home and family circle, and if the child was separated from this environment, to appreciate what effects such separation would have upon the child. If there were problems of behaviour he had to be able to advise the mother on their management. Dr. Collins asked whether undergraduates (and graduates) were adequately trained in these aspects of child care. At the end of his training what knowledge had the undergraduate or pædiatric resident medical officer, for instance, on the management of the pre-school child? Was he able to advise the mother upon the many problems she met in handling her pre-school child—a period in life when good management was now regarded as so important for the development of a stable and well-adjusted personality? His knowledge, they all knew, was pitiful compared with that of a pre-school teacher.

Apart from the failure of pædiatricians to adjust the teaching of the medical student to the changing patterns of disease and society mentioned by Dr. Clements, there were two other factors which had reduced opportunities for teaching the various aspects of total needs of the child. Firstly, there was the changing function of hospitals in which most teaching took place. With modern medicine and changes in society, the teaching hospital was becoming much more a centre for the investigation of difficult problems and rare diseases; there was less opportunity to see common problems. This could mean a concentration upon problems and detail often irrelevant to a well-balanced training. The second change which had occurred over the last few decades had been the gradual disappearance of the type of teacher or consultant who was a general practitioner, or who had been in general practice at one stage of his career. Such men had been in touch with community problems and

were able to teach with perspective. The teachers in all hospitals had become more and more specialized, and such teachers themselves often emphasized technical detail at the expense of the broad background of the personality of the patient and his social background.

These two defects in hospital teaching were being met to some extent by allotting the student to a general practitioner for a period during his course. This was now done with considerable success in Melbourne where the student spent one or two weeks with a general practitioner. In certain British schools, too, this practice had become a routine part of the curriculum. In the future, it would seem advisable for the student to spend an even longer time with a general practitioner if suitable arrangements for teaching could be made.

In view of all these changes, their first question might concern the length of time allowed in the medical curriculum for the teaching of pædiatrics. Was it adequate? In most centres Dr. Collins thought that they could now say that it was a reasonable period. If so, then there were two further questions which they should ask. Firstly, in the time available, how should they organize teaching so that the student obtained a satisfactory knowledge of the total needs of children—particularly a knowledge of psychological factors and social factors, and a knowledge of growth and development? Secondly, if they were not satisfied with the possibility of adequate teaching, was there any way in which the medical curriculum should be revised to provide opportunities for more adequate training?

Firstly, there seemed no doubt that teaching might be organized in a variety of ways to provide better teaching upon the total needs of children.

1. In the first place all clinical teaching should be directed towards regarding the patient as an individual rather than as a case of a particular disease. This sounded obvious, but required emphasis. With the in-patient, for instance, apart from a discussion of the diagnosis and treatment of the major lesion which caused hospitalization, it should be possible to discuss his development and mental and physical achievements, his reaction to hospitalization and how any ill-effects were diminished, his reaction to illness, the social background—with perhaps a social worker's report or the social worker herself present, the advice which would be given to the mother, the method of managing him when he was discharged, the community services which might be used, and the relationship to the general practitioner.

2. In regard to out-patient care, Dr. Collins said that the student could learn a great deal about total needs in the out-patient department if the teacher set out to show how a good consultation was carried out. He could learn much concerning the art of medicine, about the method of taking a history which would reveal the child's problems, development and personality, and about the management of both patient and parent. There were two essentials in making such teaching first class. Firstly, the number of students should be small so that an atmosphere of intimacy was present—Professor Spence suggested no more than two or possibly three students; and, secondly, the teacher should be experienced and have adequate time to demonstrate the art of medicine at this level. This meant that the teacher should not be burdened with a large number of patients in the out-patient clinic, but that he should be provided with just the number and type of patient he required for his teaching purposes and have no other responsibilities.

3. Discussing the creation of an atmosphere of appreciation of the total needs of children, Dr. Collins said that in the hospital where the student worked there should exist an atmosphere of sensitivity to the total needs of children. This meant that all members of the team associated with the care of children, and particularly the ward sister and nursing staff, should be aware of the emotional and physical needs of children. It meant the welding of medical, nursing and occupational staff and social workers into a team caring for children. The presence of mothers in the ward, visiting and caring for their child, with a frank and friendly relationship between them and the hospital staff, was essential for this atmosphere.

4. In regard to undergraduates and parent interviewing, it was unfortunate that the undergraduate in the ward was rarely present when the mother of the child admitted to hospital was first interviewed by the resident medical officer, for it was difficult for anyone to appreciate the intimate details of a child and his illness without this initial contact. It seemed essential that the undergraduate should be present when the resident was taking the history from the parent, so that he could take his own notes and ask further questions if he so desired.

5. Dr. Collins referred to special training in growth and development, psychological factors and community factors. He said that demonstrations and lectures on these subjects, visits to pre-school centres, such as the Lady Gowrie centre, and attendance at infant welfare centres, all played a part in training in these fields and appeared to have been used increasingly in all teaching centres. Well-planned lecture-demonstrations achieved something in educating, but it appeared essential for the student to play a more active role—for there to be more student participation—if he was to learn effectively. Instead of a demonstration and discussion upon a pre-school child, for instance, the student should be asked to make a longer case study of such a child—along the lines which, Dr. Collins understood, were being adopted in Sydney and Brisbane at the Lady Gowrie centres. There the student observed his child for two mornings, noted behaviour and development, examined the child on the third morning, studied the social and medical records at the centre, made a home visit, and on the fourth morning presented a report to his group covering physical, social and emotional development and took part in the group discussion upon the problems. This took time and well-trained staff to lead discussions.

In pædiatric teaching, however, they were all aware of the difficulty of arousing the interest of medical students in what, to them, were such refinements of medical experience. So much of preventive medicine, the psychological aspects of pædiatrics, and problems of growth and development, appeared trivial and unimportant to many medical students at this stage of their career. They had already become conditioned to the importance of "bigger things"—gross pathological lesions, malignant disease, for instance, and the great surgical skill required to excise the offending portion of human anatomy (though they might learn little of the effects of such heroic surgery upon the few remaining years of the patient's life). Unfortunately, too, they had to influence those who had taken up medicine because it was a lucrative profession and were interested mainly in passing examinations for that purpose. During the post-war years when often at least one student in a group was a parent, it had sometimes been easy to rouse the interest of the group in these aspects of child care, but in future, with few married students, the prospects of obtaining the desired amount of interest were more remote, and it was here that Dr. Collins returned to his second question. Even if they did everything mentioned, were they, as pædiatricians, satisfied with the possibility of adequate teaching to undergraduates in the total needs of children? Could anything more be done to make the students more receptive to their pædiatric teaching? Personally, he thought that more could be done, because the student had been wrongly or inadequately conditioned in his pre-clinical years. Should they be satisfied with a medical curriculum which had not changed much in decades, a curriculum which was arranged to recapitulate the history of medicine in much the same way as the fœtus in its development recapitulated the history of the human race, which directed the student's mind to technical aspects of disease during his pre-clinical years and failed to emphasize early in the course that doctors were being trained to prevent disease?

The average student entered the medical course with idealism, an interest in human nature and the hope that he could improve the health of the community. Yet he was given no opportunity during his pre-clinical years to maintain his interest in, or increase his knowledge of, the human individual and his reaction to environment or sickness. The student's perspective of his future functions was distorted during the pre-clinical scientific period, yet surely they should aim at maintaining his perspective from the first pre-clinical year. It would seem desirable that as early as possible in his medical training the student should be introduced to the concepts of human ecology. He should learn something of the structure of society, of man's reaction to his environment, aspects of mental health and perhaps anthropology. Why should a student not attend a prenatal clinic and hear and take part in the prenatal management of a patient and follow the condition through in his first pre-clinical year? This could be an introduction to preventive medicine, the study of nutrition, an interest in embryology, and community services assisting the mother and later her infant. He might even be attached to this family and follow them throughout his medical course, as was being done in some American centres. He might see a patient with diabetes or poliomyelitis and appreciate early the effect these illnesses had had upon the patient's life or on his capacity to earn a living, and at the same time receive a stimulus to the study of physiology or the anatomy of the muscular system. Other illustrations could be given. Surely if such changes were introduced and the student

received this human and sociological interest from the beginning, pædiatricians would find it much easier to interest him in the pædiatric course when he reached them.

Such changes appeared essential for patient care in all age groups, as well as those including children, if patients were to be treated as individuals and the student was to think about medicine as a whole. This attitude had provoked a number of experiments in the United States of America, in which those present should be interested. In two centres a complete reorganization of the medical curriculum had taken place and in a third new centre they were starting with new concepts.

In Denver, which Dr. Collins had visited in 1951, a new curriculum had been started at the University of Colorado in 1947. Washburn's influence, following his excellent pædiatric studies in growth and development over the previous twenty years, had played a big part in the changes. A 10% cut in the time allotted to the pre-clinical sciences had been made to permit introduction of integrating courses such as Washburn's "Medicine as Human Biology", in which a course of lectures and demonstrations was commenced in the first term of the first pre-clinical year. The main theme was growth and adaptation; the student was introduced to a variety of patients illustrating various aspects, and to community services.

At the Western Reserve University School of Medicine, Cleveland, the Commonwealth Fund had provided 300,000 to 400,000 dollars annually for the working out and putting into effect over a period of five years of an entirely new medical curriculum, remaking the teaching programme from the ground up in an integrated manner. Patients were presented early in the course and frequently thereafter to help students learn how to appraise an individual, to illustrate problems studied in the basic sciences, and to show the impact of illness both on individuals and on the community.

At the new University of California Los Angeles Medical School the buildings had been planned to arrange for a better integration of clinical and pre-clinical work, and there was an early introduction of patients and sociological aspects.

Dr. Collins then discussed post-graduate medical training. He said that two main problems arose—firstly, the need for more opportunity for training of graduates at the resident level, and, secondly, the need for improving the training of "residents" in the total needs of children.

They could anticipate that most graduates would be general practitioners doing some pædiatric work, yet in Sydney and Melbourne only 8% to 10% obtained resident medical officer training. The best graduates obtained these positions, yet from a community aspect it was the poorer medical graduates who were most in need of supervised pædiatric training. Could more graduates be given resident experience for shorter periods without the lowering of standards? Could some receive training and experience with responsibility in casualty departments and general clinics? This would require more space, and more intensive training by sound pædiatricians, but Dr. Collins thought that they should face that problem.

How could resident training in an appreciation of the total needs of children be improved? In the United States of America there was a conscious attempt to provide educational facilities for resident medical officers beyond those routinely provided in ward patient care and attendances at conferences and clinico-pathological meetings, and, of course, there were there many more conferences in the way of "rounds" and "grand rounds". Should not all "residents" have a period of responsibility in caring for well children at infant welfare centres and at pre-school centres, or with behaviour problems in a psychiatric clinic? Should the "resident" spend more time at a pre-school centre acting as a kindergartener, along the lines put forward by Mercer, who suggested that the "resident" would learn much more rapidly if he was emotionally involved in various situations with the pre-school child? Should not a course of lectures be available in child development and child psychology which the "resident" and perhaps other groups—ward sisters, social workers, occupational staff—might attend as post-graduates?

In regard to other workers there was the need for improving facilities for the training of other groups, such as trained nurses, social workers and occupational therapists at a post-graduate level in the total needs of children. Dr. Collins said that in his survey he had mentioned the need for a more extensive training of infant welfare sisters, particularly in the management of the pre-school child and the behaviour problems about which they would be asked for advice from the mother. A broader training generally was advisable for nurses and infant welfare sisters along the lines mentioned for medical students.

Discussing teachers, Dr. Collins said that without knowing a great deal more about the training of teachers, it would be presumptuous for him to comment upon their education in the total needs of children. Their problems, he understood, resembled the medical problems in that there was insufficient teaching upon the psychological aspects of child development and the emotional needs of the child. When a child was behaving badly in school, for instance, the well-trained teacher would often recognize that this was a reaction to an unsatisfactory family situation and deal with it by discussion with the child or parents or by referral.

In regard to parent education, Dr. Collins said that the need for it had been stressed by Dr. Jackson. If the various groups had been well trained in the total needs of children, personal advice and counselling, whether from physician, infant welfare sister, pre-school teacher, or school teacher, should be regarded as one of the most effective means of educating the parent. It was essential that the pædiatrician or family doctor should devote adequate time to meeting the parents' questions and answering them frankly in the course of ordinary medical practice, if he was to play his part in preventive medicine.

Group discussions were a most effective means of parent education, and when well conducted had been considered more effective than any other means of parent education. There was a great field for development in group discussions. There were active groups at some pre-school centres, especially the Lady Gowrie centres. A small number of schools had regular parents' meetings for educational purposes, but even with the major public schools it was surprising how few held such meetings, though they had been shown to be popular and to meet a great need.

Many other techniques for parent education could be used once centres for group activity were available—lectures, parents' news sheets, library facilities, posters, films, and opportunities to participate in other activities of the centre.

There was much more that could be said. Pædiatricians were left with the impression of the need for more facilities for adequate teaching in their centres, the need for more teaching staff, and the need for well-trained teachers who had been better trained in the total needs of children than many pædiatricians had been.

Discussion.

Dr. Ralph H. Crisp (Perth), opening the discussion, said that, as might be expected in a vast State, in which the pioneering stage of development could still be seen quite close to the cities, some aspects of child health were not well provided for. The lack of a medical school in Perth was one serious disability.

When the bulk of a population existed in a few widely separated towns it was natural that the children in the capital city were first served. This geographical fact was, of course, not applicable only to Western Australia. Future policy envisaged some measure of decentralization of medical services whereby the State would be zoned, and base hospitals developed at strategic points. Child health and other services would ultimately be brought to these centres. It would be a long time, however, before this vision became a reality. Till then the handicapped child had to be brought to town and this could be a severe and costly business. Days of travel by boat, hours by plane, or weary miles by rail or road—all could cost many pounds and limit the accessibility of the service. Travel concessions could be extended. Dr. Crisp also emphasized the great need for cheap hostel accommodation near the main clinics for those requiring long-term treatment. Such hostels had the additional virtue of increasing the efficiency of the available hospital beds. "Out-back" children were ill served at present and it was well that their needs should be kept in perspective. It was, of course, right and proper that advances should be made in the major centres to the limit of what was possible, but, at the same time, every assistance should be extended to under-privileged groups and districts. Development in remote areas was being hampered by the lack of, and great cost of, adequate medical services for the family.

The native child posed a disturbing problem in Western Australia. In the south-western districts half and quarter castes, and near whites, were breeding at a rate 50% higher than that of the white population. The children of these folk along with the offspring of "problem-families" anywhere were unlikely to derive much benefit from routine services, as their parents were very often not particularly intelligent.

nor keen and cooperative in seeking the best available for them. It was said that native children took education very well up to the age of about twelve to fourteen years, but that they subsequently lost their drive and became erratic and unreliable. This was about the age at which they became aware that there was no place for them in the community. A lot could be said about the "total needs" of these groups, but this would take a special chapter. Some time the challenge would have to be accepted of bringing to them the full benefits of child health.

The State reports should be regarded as first drafts only, requiring close scrutiny and coordination. The stock-taking meant a lot of work, but had not been boring as feared. On the contrary, it had been an interesting and stimulating exercise. The knowledge accumulated and summarized, together with the deliberations of the present session, made a volume of most valuable material which could be made available, when called for, for presentation to the public and for the guardians of public health and the public purse. In conclusion, Dr. Crisp congratulated Dr. Jackson on the results of his "quiz". It had put the Australian Pædiatric Association into a position from which it could show authority, exercise leadership, and perhaps do a service of inestimable value to the community.

Dr. R. Wall (Launceston) pointed out the danger of misdirection of effort and resources which could result from emotionalism. This, he said, often led to the intrusion of subnormal children into institutions intended for children who were mentally normal though physically handicapped, and led inevitably to a falling-off in the benefits derived by the children for whom the institutions were primarily intended.

Dr. A. J. Metcalfe (Canberra) said that nothing could be done about any problem until facts were available and he applauded the action of the Australian Pædiatric Association in instituting this survey. His department would now know that it could look to the Association for authoritative advice on child health matters, and he considered the Association would thereby be making a valuable contribution to national welfare.

Dr. Metcalfe also mentioned leprosy among natives which was spreading, especially among children.

Dr. B. Meredith (Melbourne) spoke of the work of the Victorian infant welfare centres, and said that much of the parent guidance to date had been given by the infant welfare sisters. The scope of this advice was now expanding to include advice on pre-school children and emotional and social problems. For this reason the training had had to be revised, and the sisters now had some post-graduate instruction at the Kindergarten Training College.

There were three other avenues of parent guidance that her department had developed—(*a*) in ante-natal clinics, (*b*) by correspondence scheme, (*c*) by the distribution of literature, which, judging by the demand, was a fertile field for development.

Dr. L. Hughes (Sydney) spoke of the rheumatic child which he termed the Cinderella of the handicapped. Recovery hospitals in England had done valuable work for years, although recently some criticism of the kind mentioned by Dr. Sims had been made. Nevertheless a recovery home in Sydney had filled a useful role. Rheumatism was becoming more prevalent, and the Smith Family Home was being enlarged.

Dr. Hughes emphasized the necessity for the avoidance of "heart consciousness" among rheumatic subjects.

Dr. S. Williams (Melbourne), referring to Dr. Meredith's remarks, recommended further extension of the work along the present lines. He applauded the sentiments expressed in the opening papers, but urged that social and psychological considerations should not be allowed to dominate the primary object in practice and teaching, namely, the treatment of the sick.

He hoped that work on the survey would continue and recommended that a further report should be given at the next meeting.

Dr. J. Colebatch (Melbourne) stressed the importance of the education of medical teachers—consultants and "residents". At the same time the students were still the most important, and they should grow up thinking that the social side was as important as anatomy, physiology and clinical medicine. He advocated the earlier introduction of social teaching into the medical course, and suggested two possible means—(*a*) appointment of a director of medical studies, (*b*) institution of a chair of social medicine.

Dr. F. Arden (Brisbane) thought that students were influenced by examination questions and that social and environmental factors might be introduced into these. He also suggested that a hospital's services might be extended to patients in their homes after discharge, and this would give resident medical officers and students an insight into home background.

Dr. H. Williams (Melbourne) said that in Melbourne some students had an opportunity of visiting their patients at home in company with an almoner who had also a nursing training.

Professor L. Dods (Sydney) mentioned Sir James Spence's use of the word "understanding". An essential part of a consultation was explanation, which was impossible without understanding. How was understanding to be taught to students? Certainly not by didactic methods, but possibly a start could be made by taking students to the home.

Dr. R. Southby (Melbourne) spoke in support of Dr. Collins's ideas of education.

Dr. K. Brennan (Melbourne) said that the choice of this subject for a round table discussion was most stimulating to a public health worker. The community should look to the Australian Pædiatric Association for a lead and he hoped it would always be ready to give that lead lest it be given instead by those unqualified for the task.

Dr. Brennan then referred to the suggested enlargement of the course for infant welfare sisters, and thought the instruction would have to be very carefully given in order to avoid equipping nurses with information that they might possibly pass on in a dangerous and misleading form.

Dr. F. W. Clements, from the chair, then suggested that the discussion might be directed towards the role and training of the nurse. He had been appalled by the nurses' lack of knowledge of the right sort. When they appreciated this lack, nurses themselves blamed it on to their basic training. He thought that nurses should "exude the atmosphere" in which it was hoped to train students.

Dr. V. Collins (Melbourne) considered that the medical profession did not take sufficient interest in the teaching of nurses.

Dr. R. Green (Sydney) said that the basic training of nurses in Australia varied from State to State. If a uniform Australian system of training could be drawn up, it might include some social and preventive aspects.

Dr. N. Wettenhall (Melbourne) drew attention to a central school for nurses established in Melbourne to which some nurses were sent from all hospitals. Pædiatrics, including emotional development and care, was part of their course. Dr. Wettenhall also stressed the need for pointing out to the student at the outset that one of the reasons for his attending a children's hospital was to learn the preventive side of pædiatrics.

Dr. M. Powell (Melbourne) said that he felt sure there was scope in each State for an institution to deal with hopeless cases.

Congenital Thrombocytopenia.

Dr. S. E. L. Stening read a paper entitled "Congenital Thrombocytopenia" (see page 210).

Dr. J. H. Colebatch (Melbourne) said that Dr. Stening's paper had been particularly valuable in drawing attention to a condition which text-books implied was very rare. Dr. Colebatch agreed that it must be less uncommon than had been supposed. He knew of one case in the Melbourne Children's Hospital, and in the past two years he had treated three other patients. The first child was born with a generalized eruption of petechiæ which rapidly increased. At four hours the blood contained 12·9 grammes *per centum* of hæmoglobin and 1632 nucleated red cells per cubic millimetre; the platelets were "extremely scanty", and a Coombs test gave a negative result. Despite blood transfusion the patient died within twelve hours. There was no family history of purpura; there were two siblings, one a spastic. The mother, however, had been given desensitizing injections for hay fever until the seventh month. At eight months she had another injection which caused a severe anaphylactic reaction, followed by nausea and anorexia till term. She had no purpura, but had a large retroplacental clot at delivery. The frequent association of allergy with thrombocytopenia suggested an ætiological relationship in this case. The mother had since had another pregnancy without desensitization and this child was normal.

The second child at eight weeks of age had suddenly developed severe thrombocytopenic purpura, with 9000 platelets per cubic millimetre, hæmoglobin 8·7 grammes *per centum*, and an active marrow containing numerous megakaryocytes of which less than 10% were normal. She had

gained weight well throughout, had had at three weeks a successful B.C.G. vaccination, and at six weeks was treated for lack of sleep, splenomegaly and mild upper respiratory tract infection. The mother had suffered from rheumatoid arthritis and from a hæmorrhagic state several years previously. The child was given cortisone and blood transfusion, and was now well. Dr. Colebatch thought that one might speculate on the relationship between her thrombocytopenic crisis and the mother's constitutional disorders. These two cases might qualify for Group III of Dr. Stening's classification, or perhaps they belonged to Group I.

The third child was a freak case. On her second day, over the left breast was noticed a bruise which grew into what seemed a large firm hæmatoma, and was followed by hæmatemesis and bleeding from the umbilicus. The "hæmatoma" steadily enlarged. At five weeks she had a prolonged bleeding time, poor clot retraction and very scanty platelets, rising to 130,000 per cubic millimetre after blood transfusion. The father and paternal grandmother both had a history of bleeding. Not until the age of two months was the "hæmatoma" shown by biopsy to be an anaplastic sarcomatous neoplasm, to which the thrombocytopenia was apparently due.

Recent Work on Cœliac Disease.

Dr. Charlotte Anderson (Melbourne), a visitor, introduced by Dr. Howard Williams, read a paper entitled "Recent Work on Cœliac Disease" (see page 211).

Dr. P. L. Hipsley (Sydney) asked whether gluten had been investigated further to identify the specific toxic fraction.

Dr. Anderson replied that some further experiment had been carried out, but the specific toxic fraction had not yet been identified. Gluten had been hydrolysed to amino acids by acid hydrolysis and this hydrolysate found to be inactive.

Dr. S. E. L. Robertson (Sydney) said that he would like to emphasize what Dr. Anderson had said about locally manufactured cornflour. He had found that much of the cornflour sold was made from wheat, and that it would be advisable to find who did prepare real cornflour and wheat starch, free of gluten.

Dr. M. C. Powell (Melbourne) asked Dr. Anderson if she thought that there were mild clinical grades of cœliac disease.

Dr. Anderson said that she thought there probably were, and felt certain that some of the iron-resistant anæmias of young children belonged to this group.

Dr. C. W. G. Lee (Sydney) was pleased to hear Dr. Anderson mention anæmia. He had seen three children recently suffering from anæmia which did not respond to iron, with minimal symptoms apart from abdominal distension. They had responded rapidly to a gluten-free diet.

Mercury Excretion in Patients with Pink Disease.

Dr. F. W. Clements (Sydney) read a paper entitled "Mercury Excretion in Patients with Pink Disease" (see page 213).

Professor Lorimer Dods asked Dr. Clements about the interpretation of the results of urine tests. He said that some children took teething powders, but had no mercury in their urine. He wondered how often chronic syndrome pink disease was found in Australia; he had seen three or four patients who had excessive mercury in the urine. He said he was not convinced that pink disease was caused by mercury.

Dr. H. Williams asked what effect BAL had on the patient who was treated.

Dr. S. E. J. Robertson (Sydney) said that it was a very unfortunate story. It was a case of a child with severe pink disease who sweated very badly. BAL was given with some improvement. After four days' therapy the child developed bronchitis, and BAL administration was immediately stopped. Two days later the child suddenly had peripheral circulatory failure, and died in three hours. Dr. Robertson did not think that BAL was to blame. It had been stopped for fifty-four hours before the child died. He thought that death was due to very severe pink disease.

Dr. Kate Campbell (Melbourne) said that teething powders were used by a third of a million babies in Victoria.

Dr. F. W. Clements (Sydney) said that he had followed up eighteen patients. After periods of one to three months all were perfectly normal healthy infants.

Coxsackie Virus Infections in Australia.

Mr. N. F. Stanley (Sydney), a visitor introduced by the President, read a paper entitled "Coxsackie Virus Infections in Australia, with Special Reference to Their Epidemiology" (see page 216).

Dr. S. Fazekas de St. Groth (Australian National University, Canberra) said that there were two points about these viruses which deserved mention. The first was that at the moment well over a dozen of them were lumped together under the common name of Coxsackie—this notwithstanding that they caused well-defined and distinct clinical pictures, differed in size, host range, antigenic composition, resistance to chemical agents *et cetera*. It was granted that all of them would grow in suckling mice, but by this token they might as well include the viruses of herpes, smallpox, lymphocytic choriomeningitis, yellow fever, and several others in the group. It would be sounder to call the respective strains "Herpangina Virus", "Pleurodynia Virus", "Sydney Encephalitis Virus"—or if they liked to refer to the host range of the type discovered by Mr. Stanley, perhaps "Summer Encephalitis Virus of Young Maidens". In any case, Dr. de St. Groth felt that they should prefer a logical and informative name to one which, though historical, was likely to prejudice their understanding of the true relationship between the viruses.

The other point was that these viruses were essentially benign, and it was significant in this respect that there was not a single autopsy report he could recall in the extensive literature that had accumulated since their discovery. This was of great practical importance, as the diagnosis of Coxsackie infection automatically meant good prognosis, whatever the present condition might be, and recovery without any serious sequelæ. In this connexion Dr. de St. Groth could remember a very striking instance last year in Melbourne, and perhaps Dr. Powell might be good enough to tell them about it.

Tissue Culture of Poliomyelitis Virus.

Mr. J. Thayer (Melbourne), a visitor introduced by Dr. Stanley Williams, read a paper entitled "Tissue Culture of Poliomyelitis Virus" (see page 215).

Dr. S. Fazekas de St. Groth (Australian National University, Canberra) said that no one could fail to be impressed by the great advances which the introduction of the technique meant. If it would not do more than establish or confirm the diagnosis of poliomyelitis, its place in the routine of the virus diagnostic laboratory would be ensured. But it did much more than that. It enabled research into poliomyelitis to be carried out on a scale inconceivable earlier, and already the very first results were of extreme importance. It could be shown that the virus regularly appeared in the blood during the early stages of infection, and this viræmia was a standard feature of the disease. Thus poliomyelitis ceased to be a pathological entity of its own, but rather fell into step with the rest of infectious diseases of long incubation period, following the pattern revealed by Professor Fenner. And this was not just a nice distinction at the theoretical level. It meant that they should be able to stop the disease half-way, before it could enter the central nervous system, either by passive immunization as was currently being tried in the United States of America, or by active immunization, which Dr. de St. Groth regarded as the more hopeful approach.

Here, again, the tissue culture technique came to their aid, as it was now practicable to obtain virus in unlimited quantities for the production of killed vaccines; also, the way was open to develop an attenuated line of virus suitable as live vaccine. This development, which had been completely successful in the case of smallpox and yellow fever, was definitely on the cards.

The scope of the method was, however, much wider than the field of poliomyelitis. There were several diseases of known or suspect viral ætiology still beyond reach, simply because it was not possible to grow the virus in the laboratory. There was a fair chance that some of these could be established on in-vitro cultures of the specific tissue which served as their natural habitat. And from then on it was only a short step to producing a vaccine and combating the disease.

Thus, instead of criticizing the paper, Dr. de St. Groth made a plea that whenever a virus diagnostic service was established in a hospital, facilities should be provided for the technique in question, not only as a method of routine diagnosis, but as one of the most promising lines of research into infectious diseases.

Dr. A. A. Ferris (Melbourne) said that the Commonwealth Serum Laboratory tissue culture unit had been established to develop techniques applicable to poliomyelitis, and was not concerned with laboratory diagnosis; at the same time one approach in the diagnostic field had been made in an endeavour to sort out some of the non-paralytic poliomyelitis cases. At the moment they saw perhaps 50 such patients per week at Fairfield Hospital. These patients presented with neck and spine stiffness and pleocytosis and raised protein content were found in the cerebro-spinal fluid. Fecal specimens and serum samples (one early and one late in the disease) had been collected from a series of patients. In a small number of cases poliomyelitis virus was isolated from the fæces, but it had not been possible to demonstrate a rise in antibody titre in any instance. Apparently antibody was present in considerable titre very early in the disease and persisted in maximal titres for many months. The findings, therefore, were disappointing.

Mr. Thayer had told them that in Melbourne during 1951 type I virus had been the predominant type with a few type III strains, whereas since the middle of 1952 until the present time there had been a different picture—no type I strains had been encountered, and type II was predominant with a few type III strains still occurring. Mild epidemic conditions prevailed in Melbourne at present and it might be worth noting that an unusually high proportion of the current cases were of mild bulbar poliomyelitis—for a time 50% of patients presented with bulbar poliomyelitis. During 1951, whilst type I was the epidemic strain, perhaps 10% of patients had suffered from bulbar poliomyelitis.

Mr. Thayer had stated the opinion of Dr. Salk's laboratory in Pittsburg when referring to the possibility of a poliomyelitis vaccine. These American workers found that monkey kidney tissue gave higher viral titres than did monkey testicular tissue and tended to favour a formalinized vaccine. It was worth noting that this conflicted with the view expressed by Professor Sir MacFarlane Burnet. Burnet believed that a live attenuated vaccine, probably given under cover of γ globulin, might ultimately prove the method of choice. A vast amount of careful experimental work with an extensive clinical trial remained to be done before the problem of an effective and safe poliomyelitis vaccine was solved.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

THE MEDICAL SOCIETY OF VICTORIA.[1]

LIST OF MEMBERS, JANUARY, 1868.

Executive.

President: Dr. Neild.
Vice-Presidents: Dr. Bird and Dr. Jonasson.
Treasurer: Dr. Fetherston.
Hon. Secretary: Mr. Blair.
Hon. Librarian: Dr. Nicholls.
Ordinary Members of Committee: Mr. Gilbee, Dr. Black, Dr. Graham, Dr. McCarthy.

Members.

Barker, Edward, F.R.C.S., M.D., Melbourne; Barrett, James, M.R.C.S. and L.S.A.; Bayldon, John, M.B. (Lond.), L.R.C.S. (Ed.), L.R.C.P. (Ed.); Bird, Samuel Dougan, M.D. Stand. and Melb., M.R.C.S., L.R.C.P., L.S.A.; Black, Joseph, M.R.C.S., M.D. (Melb.); Blair, John, L.R.C.S. (Ed.); Bower, Thomas Aubrey, M.R.C.S. and L.K. and Q.C.P.I.; Bragge, John de la Roche, M.R.C.S. and L.S.A.; Cutts, William Henry, M.D. (Ed. and Melb.), L.S.A.; Day, John (Geelong), M.D. (Glas.), M.R.C.S.; Fetherston, Gerald Henry, M.D. (Melb.), L.R.C.S. (Ed.), L.F.P.S.G. and L.A.H.D.; Fletcher, Edward, M.R.C.S.; Gilbee, William, M.R.C.S.; Girdlestone, Thard Mountain, F.R.C.S.; Graham, George, M.D. (Melb.), M.R.C.S.; Gray, Andrew Sexton, M.R.C.S. (Eng.); Haig, William, M.D. (Maryland, U.S.A.); Jonasson, Herman, M.D. (Wurzburg and Melb.); Lempriere, Chas. Monteiro D'Almeida, M.R.C.S.; Lilienfield, Bernhard, M.D. (Marburg and Melb.); Martin, Lawrence Joseph, M.D. (Melb.), L.R.C.S. (Ed.), L.A.H.D.; McCarthy, Chas., M.D. (Melb.), and L.F.P.S.G.; Molloy, William Thomas (Balmoral), L.K. and Q.C.P.I., L.R.C.S. (Ed.), L.A.H.D.; Motherwell, James Bridgeham, M.D. (Glas. and Melb.), L.R.C.S.I.; Mousse, Antoine, M.D. (France and Melb.); Neild, James Edward, M.D. (Melb.), L.S.A.; Nicholls, Austin Hinsson, L.R.C.P. (Ed.), L.R.C.S. (Ed.); Pincott, Rupert (Geelong), M.R.C.S. and L.S.A.; Rankin, William Bailey, L.R.C.S. (Ed.); Rudall, James Thomas, F.R.C.S.; Thomas, David John, M.D. (Stand. and Melb.), F.R.C.S., L.S.A.; Tracy, Richard Thomas, M.D. (Glas. and Melb.), L.R.C.S.I.; Van Hemert, Francis Thomas, M.R.C.S., L.S.A.; Wilkins, John, F.R.C.S.; Wooldrige, Henry, M.R.C.S. and L.S.A.; Woolhouse, Henry Hervé (Gisbourne), M.R.C.S.

Honorary Members.

Bleasdale, Rev. J., D.D.; Ellery, Mr., Govt. Astronomer; Ralph, Mr., M.R.C.S., L.S.A.; Mueller, Dr., F.R.S., Govt. Botanist.

[1] From the original in the Mitchell Library, Sydney.

Correspondence.

CHOLEDOCHODUODENOSTOMY.

SIR: Mr. Starr states in his letter that the only reason which he could advance for the absence of cholangitis (and recurrent obstruction) was technical. I believe a further and important reason lies in one great factor, that is, in the part of the gastro-intestinal tract chosen for the anastomosis. The duodenum and the jejunum can be regarded as empty bowel in the sense that any contents are always passing distally. The stomach has contents retained and at times under tension. I believe that cholecystgastrostomy will frequently result in attacks of cholangitis. There is further evidence for this to be found in attempted demonstration of the fistulous tract by barium meal. It appears that it is often difficult to make the barium enter the common bile duct, even when it has eventually been proved that a good wide stoma still exists. The stoma of a cholecystgastrostomy is much easier to demonstrate.

Yours, etc.,

L. IAN BURT.

Wickham Terrace,
Brisbane, B.17,
July 13, 1953.

THE TREATMENT OF CHRONIC RHEUMATISM WITH "BUTAZOLIDIN".

SIR: In answer to Dr. Michael Kelly's letter of July 4 and his paper of May 16. It is difficult to criticize Dr. Kelly's results that show such a small percentage of serious bi-effects when one considers the increasing number of reports of fatal and dangerous sequelæ appearing each week in British and American medical literature.

It is an accepted concept that cortisone, ACTH and "Butazolidin" suppress rheumatoid arthritis but do not cure. The condition returns when the drug is ceased. Almost all drugs that are of proved therapeutic value in rheumatoid arthritis have some objectionable bi-effect, and "Butazolidin" is certainly no exception.

Drugs which suppress rheumatoid joint symptoms are prone to encourage over-use of pathological joints with consequent deterioration; but, worse than that, the drawing of an analgesic veil over painful symptoms, which "Butazolidin" does so efficiently, tends to dislocate attention from fundamental and important treatment. Allow me to mention a few examples. Firstly, in those cases where the stress factor is predominantly infective, permanent cure can frequently be accomplished. Chronic sinus infection treated non-surgically and obscure dental sepsis are common illustrations. Secondly, the general condition of the patient, anæmia, decalcification, tissue fluid retention *et cetera* must not be overlooked on account of easy analgesia. Thirdly, the simple measures of splinting and a proper balance between remedial exercises and adequate rest are easily ignored for the same reason. Fourthly, where long-term suppression is desirable, treatment of proved value and with less hazards can with advantage be given priority over "Butazolidin". Rheumatoid arthritis is well known to be fickle in its responses, and I have found "Butazolidin" of great value in some difficult cases. Bi-effects in my comparatively small series include neutropenia, œdema, ulcerative stomatitis and hæmorrhage from the alimentary tract. Small-

dosage schedule and, except in a few cases, short-term treatments have been employed. Some rheumatoids do not appear to obtain benefit from "Butazolidin".

Osteoarthritis of the hip is treated in the first instance by ultrasonic radiation, which fails to benefit about 30%. After nearly two years' experience of the method I know of no other conservative treatment capable of giving such long periods of absence of pain, in some cases lasting more than a year before repetition of treatment is necessary. Ultrasonic radiation is simple, perfectly safe and has no bi-effects. For the ultrasonic resistant cases "Butazolidin" is prescribed for short periods of analgesia when such are necessary for long journeys and business. Doses in these cases have been higher, 0·6 to 0·8 gramme daily. There have been no bi-effects other than nausea.

In acute gout where short-term treatment is advantageous, I have found "Butazolidin" greatly superior to colchicine. Adequate safeguards must be taken to prevent crystallization of uric acid, which is excreted in great quantity.

I trust that this will stimulate further controversy on Dr. Kelly's important article.

Yours, etc.,

149 Wickham Terrace, JOHN SHANASY.
Brisbane,
July 13, 1953.

CONGENITAL HYPERTROPHIC PULMONARY STENOSIS IN ABORIGINAL CHILDREN.

SIR: Inquiry among practitioners here and in the eastern States as to whether any have seen a case of congenital hypertrophic pyloric stenosis in aboriginal children has brought negative answers. I should be very glad if anyone could enlighten me as to whether this disease has ever been found in the "full-blood" Australian aborigine. I have recently seen a case of the disease in a nine-weeks-old child of a quarter-caste aboriginal mother and a white father.

Yours, etc.,

JAMES WATSON.
Princess Margaret Hospital for Children,
Perth,
July 8, 1953.

EXCHANGE TRANSFUSION AND TRANSFUSION IN INFANTS AND YOUNG CHILDREN.

SIR: Among the list of references to an article I wrote in THE MEDICAL JOURNAL OF AUSTRALIA of May 30, 1953, entitled "Exchange Transfusion and Transfusion in Infants and Young Children", I have written Dr. P. L. Mollison's name "Mollinson". This is an error on my part which I very much regret.

Yours, etc.,

J. C. A. DIQUE.
The Brisbane and South Coast Hospitals Board,
Brisbane Base Hospitals Post Office,
Herston Road,
Brisbane.
July 21, 1953.

ANOTHER RECORD OF A HUMAN DIPHYLLOBOTHRIID IN AUSTRALIA.

SIR: It would seem that human infestation with the *Diphyllobothrium latum* group of tapeworm is not as rare in Australia as one might infer from Miss Sandars's article (M. J. AUSTRALIA, July 11, 1953), and as time passes it is likely to become less uncommon.

In July, 1952, a young Esthonian woman, resident in Australia for twelve months, presented at the Royal Perth Hospital complaining of malaise and weakness and stating that she had seen tapeworm segments in her stools at intervals since her arrival in Australia.

She was admitted, and following fasting and the administration of *Filix mas* and saline purgatives passed a complete tapeworm with scolex, which was identified by the staff of the Public Health Laboratory as *Diphyllobothrium latum*, showing the typical rosette uterine pattern. No further evidence of infestation was obtained.

Yours, etc.,

R. H. C. RISCHBIETH.
34 Church Street,
Walkerville,
South Australia.
July 18, 1953.

Post-Graduate Work.

THE MELBOURNE PERMANENT POST-GRADUATE COMMITTEE.

General Practitioners' Refresher Course in Gynæcology and Obstetrics.

A GENERAL PRACTITIONERS' refresher course in gynæcology and obstetrics will be held at the Women's Hospital, Carlton, from August 31 to September 11, 1953. The programme is as follows:

August 31: 9.30 a.m., Dr. M. Mackie, "Difficulties Associated with Induction of Labour"; 11.15 a.m., professorial unit, "Vaginal Repair" (demonstration); 2.15 p.m., Dr. W. McI. Rose, "The Role of the Physician in an Obstetrical Hospital".

September 1: 9.30 a.m., Dr. N. De Garis, "Ante-Natal Problems" (demonstration); 11.15 a.m., Dr. J. W. Johnstone, "Surgery Associated with Sterility"; 2.15 p.m., Dr. W. P. Holman, "Role of Irradiation Therapy in Gynæcological Conditions"; 4 p.m., Dr. J. Colebatch, "Neo-Natal Infections".

September 2: 9.30 a.m., Dr. J. Smibert, "Ante-Partum Hæmorrhage"; 11.15 a.m., Dr. G. B. Bearham, "Pruritus and Vulval Conditions" (demonstration); 2.15 p.m., Dr. Colin Macdonald, "Contribution of Radiology to Obstetrics and Gynæcology".

September 3: 9.30 a.m., professorial unit, "Difficulties Encountered in Cæsarean Sections"; 11.15 a.m., Dr. D. F. Lawson, "Abdominal Hysterectomy" (demonstration); 2.15 p.m., Dr. K. McCaul, "Analgesia in Labour"; 3 p.m., Dr. Gordon Ley, "Sterility".

September 4: 9.30 a.m., "Post-Partum Hæmorrhage"; 11.15 a.m., Dr. A. M. Hill, "Cancer Surgery" (demonstration); 2.15 p.m., professorial unit, obstetrical and gynæcological quiz.

September 7: 9.30 a.m., Dr. G. Simpson, "Slow Labour"; 11.15 a.m., professorial unit, "Abdominal Hysterectomy" (demonstration); 2.15 p.m., Dr. J. W. Johnstone *et alii*, obstetrical and gynæcological quiz.

September 8: 9.30 a.m., Dr. W. M. Lemmon, "Why the Breech Baby May Die"; 11.15 a.m., Dr. C. K. Churches, "Vaginal Repair" (demonstration); 2.15 p.m., Dr. G. B. Bearham and Dr. G. Simpson, obstetrical and gynæcological quiz; 4 p.m., Dr. Kate Campbell, "The Premature Infant".

September 9: 9.30 a.m., Dr. R. Rome, "Labour Ward Emergencies"; 11.15 a.m., Dr. H. B. Hattam, "Stress Incontinence Treated by Vaginal Repair"; 2.15 p.m., Dr. D. F. Lawson and Dr. W. M. Lemmon, obstetrical and gynæcological quiz.

September 10: 9.30 a.m., professorial unit, "Assessment of Disproportion"; 11.15 a.m., Dr. B. Anderson, "Endometriosis" (demonstration); 2.15 a.m., Dr. A. M. Hill and Dr. R. M. Rome, obstetrical and gynæcological quiz.

September 11: 9.30 a.m., "Use and Abuse of Forceps"; 11.15 a.m., Dr. V. E. Hollyock, gynæcological demonstration; 2.15 p.m., Dr. H. F. Bettinger, "Macroscopic Diagnosis in Gynæcological Pathology".

In addition, there will be normal ward rounds by the honorary medical staff. Residence at the hospital during the period is advised and will be available. It will be necessary for those attending to demonstrate the absence of hæmolytic organisms on throat swab examination before commencing the course. Enrolments should be made with the Secretary of the Post-Graduate Committee at 394 Albert Street as soon as possible. The tuition fee, £10 10s., is payable to the committee; the fee for residence, £6 10s., per week, is payable to the hospital.

Royal Australasian College of Surgeons.

PRIMARY EXAMINATION FOR THE F.R.A.C.S.

A PRIMARY examination in anatomy (including normal histology) and applied physiology and the principles of pathology will be conducted in Melbourne and in Sydney in November, 1953.

The written papers will begin simultaneously in both cities on Thursday, November 12, 1953. The examiners will

visit both cities for the purpose of conducting the viva-voce section of the examination.

The examination is reciprocal with the primary examinations for Fellowship of the Royal College of Surgeons of England, the Royal College of Surgeons of Edinburgh, the Royal College of Surgeons in Ireland, and the Royal Faculty of Physicians and Surgeons of Glasgow.

Each candidate will be notified by post of the result at the termination of the examination.

The examination is open to graduates of not less than one year's standing of a medical school approved by the Council of the College for the purpose.

Candidates must submit evidence of their qualification and of the date of acquirement thereof.

Forms of application for admission to the examination may be obtained from the Secretary, Royal Australasian College of Surgeons, Spring Street, Melbourne.

When entering for the examination, candidates must state whether they desire to appear before the Board of Examiners in Melbourne or in Sydney.

The fee for admission, or readmission, to the examination is £15 15s. Australian currency *plus* exchange on cheques drawn on banks outside Melbourne. The fee must be forwarded with the form of application so as to reach the Secretary at his office not later than October 1, 1953.

FINAL FELLOWSHIP EXAMINATION.

THE next meeting of the Court of Examiners for the final examination for Fellowship of the College will be held at the College in Melbourne beginning on Thursday, May 6, 1954. Candidates who desire to present themselves at this examination should apply, on the prescribed form, to the Censor-in-Chief for permission to do so on or before March 25, 1954. The appropriate forms are available from the Secretary, Royal Australasian College of Surgeons, Spring Street, Melbourne. Candidates who have already been approved by the Censor-in-Chief, but who have not yet presented for examination, may present at this examination, provided they notify the Secretary of their intention to do so by March 25, 1954. The examination fee is £21 *plus* exchange on cheques drawn on banks outside Melbourne, and must be paid to the Secretary by March 25, 1954.

The Council has decided that, as an emergency measure, one more final examination shall be held in the special branches of gynæcology and operative obstetrics, orthopædics, urology and laryngo-otology. This examination will be conducted in May, 1954. This is the last occasion on which candidates may present for the final examination for Fellowship in these special branches of surgery. After the May, 1954, examination, the final examination will be conducted in general surgery and ophthalmology only. The final examination in general surgery will be held twice in each calendar year and at least one final examination in ophthalmology will also be held in each year.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 44, of July 9, 1953.

AUSTRALIAN MILITARY FORCES.

Australian Regular Army.

Royal Australian Army Medical Corps.

The Short Service Commission granted to 1/8053 Captain (Temporary Major) A. J. Splatt for a period of one year is converted to a Short Service Commission for a period of two years.

Regular Army Special Reserve.

Royal Australian Army Medical Corps.

VX700320 Captain (provisionally) D. N. Hawkins is transferred to the Citizen Military Forces, 19th March, 1953.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED JULY 4, 1953.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	1(1)	7(5)	..	..	..	..	..	..	8
Amœbiasis	..	1	..	..	..	..	..	..	1
Ancylostomiasis	..	..	4	..	..	..	..	..	4
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	1	..	..	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	..	1(1)	7(5)	..	..	..	..	..	8
Diphtheria	17(3)	2(1)	8(2)	..	3(3)	..	..	..	30
Dysentery (Bacillary)	..	1(1)	1(1)	2(1)	1(1)	4(3)	..	..	9
Encephalitis	..	2	..	1(1)	..	..	..	..	3
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	..	1	..	..	14(5)	..	..	..	15
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	3	..	..	..	..	..	3
Malaria	..	..	..	..	4(4)	..	..	..	4
Meningococcal Infection	1(1)	8(6)	2(1)	4	..	2(1)	..	..	17
Ophthalmia	..	..	..	..	1	..	..	..	1
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	14(7)	2(2)	2(1)	4(3)	1	..	1	..	24
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella	..	24(17)	..	..	14(10)	..	..	..	38
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	17(9)	62(49)	5(2)	1	3(3)	4(3)	..	..	92
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	1	..	1(1)	..	..	..	2
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	37(27)	64(55)	21(6)	18(16)	7(2)	4	2	..	153
Typhoid Fever	1	1(1)	1(1)	..	..	..	..	..	3
Typhus (Flea-, Mite- and Tick-borne)	..	..	1	..	1	..	..	..	2
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

The notification respecting VX700320 Captain (provisionally) D. N. Hawkins, which appeared in Executive Minute No. 77 of 1953, promulgated in *Commonwealth Gazette*, No. 26, of 1953, is withdrawn.

Citizen Military Forces.

Eastern Command: Second Military District.

Royal Australian Army Medical Corps (Medical).—2/206956 Honorary Captain G. L. McDonald is appointed from the Reserve of Officers, and to be Captain (provisionally), 6th May, 1953. To be Captain (provisionally), 4th June, 1953: 2/206958 William Robert Morgan Shaw.

Southern Command: Third Military District.

Royal Australian Army Medical Corps (Medical).—The provisional ranks of the following officers are confirmed: 3/77537 Major G. G. Harkness and Captains 3/73277 J. W. Bennett, 3/101018 W. N. Sloan and 6/15358 C. S. Reid. 3/101020 Captain (provisionally) D. N. Hawkins is transferred from the Regular Army Special Reserve, 19th March, 1953, with regimental seniority in accordance with Army seniority (2nd September, 1952).

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps.

1st Military District: To be Honorary Captains.—Lucas Souvlis and Robert Eric Charles Stringer, 4th June, 1953, and Richard Simpson Paterson, 9th June, 1953.

3rd Military District.—Honorary Captain H. T. Chapman is retired, 1st June, 1953.

Medical Practice.

NATIONAL HEALTH (PENSIONERS' MEDICAL SERVICES COMMITTEES OF INQUIRY) REGULATIONS.

THE following notice was published in the *Commonwealth of Australia Gazette*, Number 47, of July 30, 1953.

I, EARLE PAGE, the Minister of State for Health, hereby give notice, in pursuance of sub-regulation (3.) of regulation 26 of the National Health (Pensioners' Medical Services Committees of Inquiry) Regulations that I have directed that Mary McDonald Thomson, of 278 Burwood-road, Burwood, in the State of New South Wales, medical practitioner, shall cease as from the twenty-first day of July, 1953, to be eligible to participate in the arrangement made by the Director-General of Health under section 7 of the *National Health Service Act*, 1948-1949, and the National Health (Medical Services to Pensioners) Regulations in relation to the provision of medical services to pensioners and their dependants and shall not again be so eligible for a period of three months after that date.

Dated this sixteenth day of July, 1953.

EARLE PAGE,
Minister of State for Health.

Australian Medical Board Proceedings.

QUEENSLAND.

THE following have been registered, pursuant to the provisions of *The Medical Acts*, 1939-1948, of Queensland, as duly qualified medical practitioners: Marr, Donald John, M.B., 1952 (Univ. Sydney); Edwards, Cecil Albert Thomas, M.B., B.S., 1946 (Univ. Melbourne); Efron, Lionel, M.B., B.S., 1953 (Univ. Melbourne); Minogue, Francis John, M.B., B.S., 1952 (Univ. Sydney); O'Hara (*née* Conley), Valda Mary, M.B., B.S., 1951 (Univ. Sydney); Marks (*née* McEwen), Betty Jean Harvard, M.B., B.S., 1948 (Univ. Sydney).

The following additional qualifications have been registered: Galea, Eugene George, M.R.A.C.P., 1953; Tonge, John Iredale, Dip. Clin. Path. (Univ. Sydney), 1951.

Deaths.

THE following deaths have occurred:

BRYDON.—Adam Gibson Brydon, on July 25, 1953, at Urunga, New South Wales.

LEWIS.—David Herbert Lewis, on July 25, 1953, at Sydney.

DAVIS.—Gilbert Maurice Davis, on July 25, 1953, at Bonegilla, Victoria.

Diary for the Month.

AUG. 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
AUG. 14.—Queensland Branch, B.M.A.: Council Meeting.
AUG. 17.—Victorian Branch, B.M.A.: Finance Subcommittee.
AUG. 18.—New South Wales Branch, B.M.A.: Medical Politics Committee.
AUG. 19.—Western Australian Branch, B.M.A.: General Meeting.
AUG. 19.—Victorian Branch, B.M.A.: Clinical Meeting.
AUG. 20.—Victorian Branch, B.M.A.: Executive of Branch Council.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £5 *per annum* within Australia and the British Commonwealth of Nations, and £6 10s. *per annum* within America and foreign countries, payable in advance.